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HEART DISEASE IN CHARITY HOSPITAL OF LOUISIANA IN NEW ORLEANS*

A SURVEY OF 8,313 ROUTINE AUTOPSIES

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A statistical analysis was made of all of the autopsies in which the deaths were attributed to heart disease at the Charity Hospital of Louisiana at New Orleans between the years 1935 to 1940 inclusive. The criteria of classification of the etiological causes of heart disease of the American Heart Association were used.††

During this period there were 19,623 deaths in the hospital of which 9,178 or 46.3 per cent were in the white race and 10,445 or 53.8 per cent were in the negro (table 1). There were 8,313 autopsies performed during this time or a percentage of 42.3 of the total deaths. Autopsies were obtained in one-third of the white deaths and 50.3 per cent of the negro deaths. Of the total number of autopsies during this period, 5,252 or 63.2 per cent were in the negro race. There were 4,957 males (59.6 per cent) of both races in the series of autopsies.

There were 1,045 autopsies (12.6 per cent of the total number) in which some type of heart disease was the primary cause of death. These varied in age from stillborn to 98 years. The negro race made up 63.6 per cent of this total number. This is almost identical to the percentage (63.2) of the negro race in the total number of autopsies.

The male sex comprised 67 per cent of the total deaths due to heart disease and 59.6 per cent of the total number of autopsies.

TABLE 1

Total Dealths, Autopsies and Cardiac Deaths at Charity Hospital at New Orleans (1935-1940)

	White Race				Negro Race				Grand
	Male	Female	Total	Per cent	Male	Female	Total	Per cent	Total
Total deaths	5729	3449	9178	46.2	6031	4414	10445	53.8	19,623
Total autopsies	1975	1086	3061	36.8	2982	2270	5252	63.2	8,313
Total Cardiac deaths	281	99	380	37.4	423	242	665	63.6	1,045

*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

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††The members of the Pathology Department of the Hospital and of the Louisiana State University School of Medicine were very cooperative, and assisted in the study of records which proved difficult to interpret.

TABLE 2

Incidence of Death due to Heart Disease

Etiology	White Race			Per	Negro Race				Grand	Per Cent
	M.	F.	Total	cent	M.	F.	Total	Cent	Total	Total
Hypertensive	101	28	129	59.4	185	109	294	69.5	423	40.5
Arteriosclerotic	96	33	127	30.5	54	34	88	40.6	217	20.7
Syphilitic	23	4	27	14.0	125	38	163	86.0	190	18.2
Rheumatic	33	17	50	42.7	34	33	67	57.3	117	11.1
Congenital	4	5	9	39.2	2	12	14	60.8	23	2.2
Toxic	8	5	13	59.1	5	4	9	40.9	22	2.1
Bacterial	9	4	13	59.1	6	3	9	40.9	22	2.1
Anemia	4	0	4	57.1	3	0	3	42.9	7	0.7
Avitaminosis	2	0	2	40.0	1	2	3	60.0	5	0.5
Combined	1	3	4	80.0	1	0	1	20.0	5	0.5
Hyperthyroidism	0	0	0	0	3	1	4	100	4	0.4
Pulmonary disease	0	0	0	0	2	2	4	100	4	0.4
Other	0	0	0	0	2	1	3	100	3	0.3
Unknown	0	0	0	0	0	2	2	100	2	0.2
Trauma	0	0	0	0	0	1	1	100	1	0.1
Total	281	99	380	36.4	423	242	665	63.6	1045	

Fifteen etiologic types of heart disease were found in this survey (table 2). As will be seen, the four most frequent types of heart disease: hypertensive, arteriosclerotic, syphilitic, and rheumatic made up 90.5 per cent of the total cardiac deaths. In this group there were 612 or 64.6 per cent in the negro race and 335 or 35.4 per cent in the white race. This compares with 63.2 per cent of negroes in the total autopsy series. These will now be discussed more in detail.

HYPERTENSIVE HEART DISEASE

There was a total of 423 deaths (40.5 per cent) due to this type of heart disease, 129 in the white race and 294 in the negro race.

White race: There were 129 or 30.5 per cent deaths in the white race (figure 1). Of this group the males made up 70.6 per cent or a ratio of 2.33 to 1. This is compared to the ratio of males to females in the routine white autopsies of 1.72 to 1. The peak of incidence of the deaths occurred at the age of 55 years and with a gradual rise starting at the age of 45 and a sharp drop after 75 years.

Negro race: A total of 294 deaths or 69.5 per cent occurred in the negro race (fig. 2). This compares favorably with the 63.2

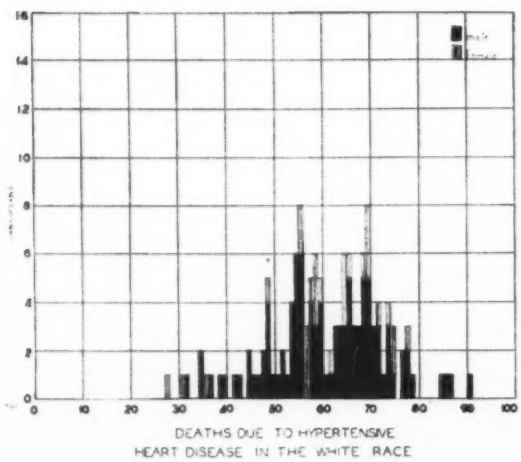

Fig. 1

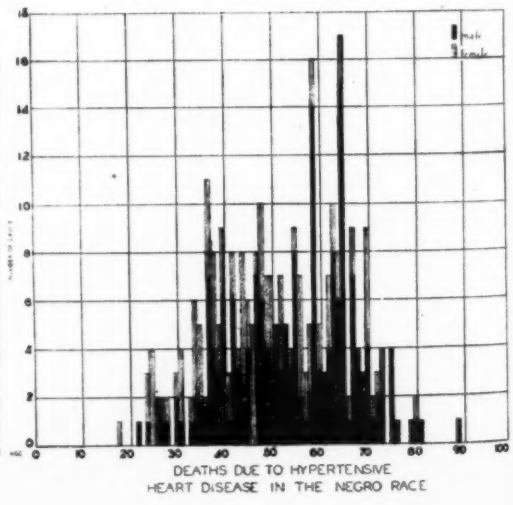

Fig. 2

per cent of the total autopsies in the negro race. The males again predominated, there being 185 or 63 per cent, a ratio of 1.58 to 1 as compared to a ratio of negro males to females in total autopsies at 1.31 to 1. There is a sharp rise in incidence at the age of 30 years, occurring about 15 years earlier than in the white race. The peak is reached at 70 years.

The most commonly associated cardiac complication in this entire group was arteriosclerosis occurring in 143 or 33.6 per cent of the cases. Rheumatic heart disease was found in 4 per cent or 17 cases, and syphilitic heart disease in 1.8 per cent or eight cases.

ARTERIOSCLEROTIC HEART DISEASE

Two hundred and seventeen deaths (20.7 per cent) were due to arteriosclerotic heart disease, 129 (59.4 per cent) in the white race and 88 (40.6 per cent) in the negro race.

White race: There were 129 deaths (59.4 per cent) in the white race which were placed in this group (fig. 3). Included in

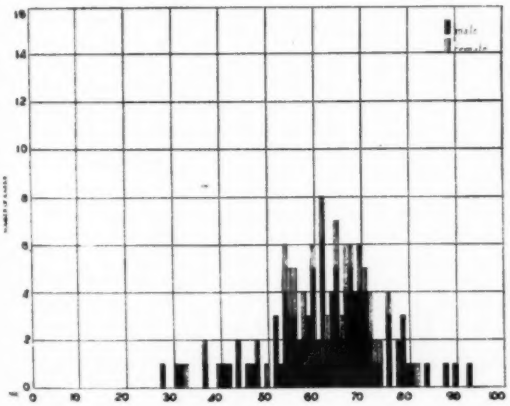

Fig. 3

this were six cases of dissecting aneurysm; five males aged 28, 52, 54, 55, and 62, and one female aged 58.

There was a total of 73 deaths due to myocardial infarction in this race, with the males predominating in a ratio of 3 to 1. In this group the males showed a sharp rise in incidence at the age of 50. This remained constant until the age of 65. Of the remaining 36 male and 14 female patients no recent evidence of infarction was found.

Negro race: There were 88 cases in this group, 54 males and 34 females (fig. 4). Of

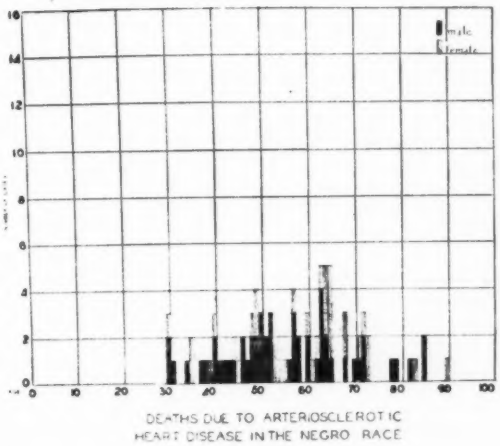

Fig. 4

the five cases of dissecting aneurysm, four were males and one female. Myocardial infarction was found in a total of 44, 27 males and 17 females or a ratio of 3 to 2. In the remainder, 23 males and 16 females, no evidence of recent infarction was found.

The complicating conditions in this group were; hypertension, 47 cases (21.6 per cent); rheumatic heart disease, 10 cases; and syphilitic heart disease, three cases.

Of particular interest are the infarctions occurring below the age of 40 years. There were six negro males, two negro females, two white males and two white females.

SYPHILITIC HEART DISEASE

A total of 190 (18.2 per cent) of the total deaths were due to syphilitic heart disease. Included in this group are 77 cases in which syphilitic aortic aneurysms were the cause of death.

White race: There were 23 white males and four white females in this group (fig. 5) or a ratio of almost 6 to 1. Of this

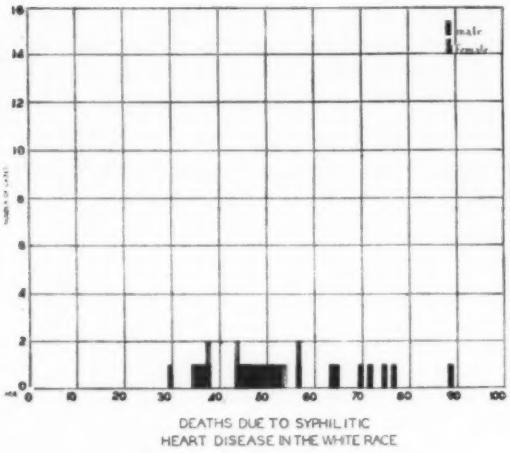

Fig. 5

number eight males and one female had large aortic aneurysms while the remainder had luetic valvulitis.

Negro race: There were 125 male and 38 female deaths occurring in the negro race, making a ratio of 3.3 to 1, males to females. There were 68 aneurysms with a ratio of five males to two females (fig. 6).

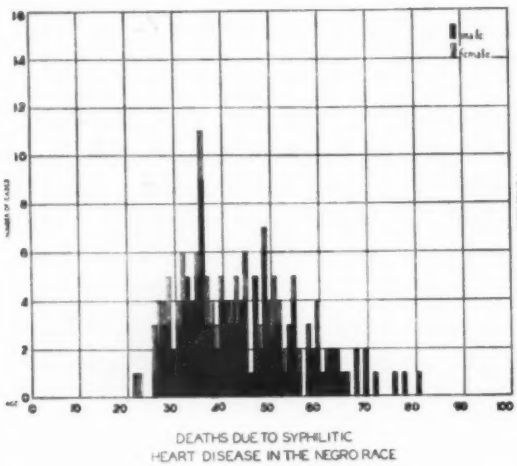

Fig. 6

Complicating cardiac conditions in the entire group of syphilitic heart disease were: rheumatic, 14 cases; arteriosclerotic, 13 cases; bacterial endocarditis of the involved aortic valve, six cases; and hypertension, five cases.

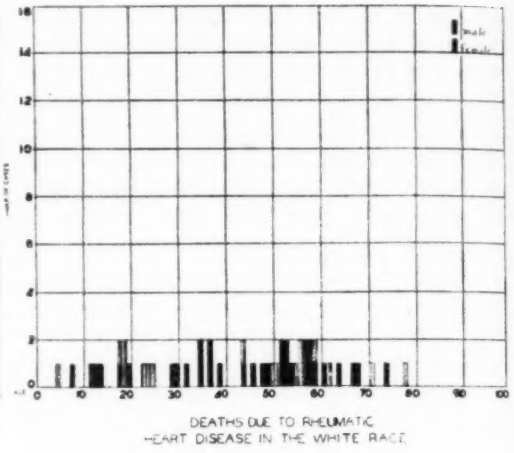

Fig. 7

RHEUMATIC HEART DISEASE

Eleven and one-tenth per cent or 117 died primarily of rheumatic heart disease.

White race: The white race made up 50 or 42.7 per cent of this group, there being 33 males and 17 females or a ratio of 2 to 1 (fig. 7).

Negro race: In the negro race the ratio was 1 to 1 with 34 males and 33 females

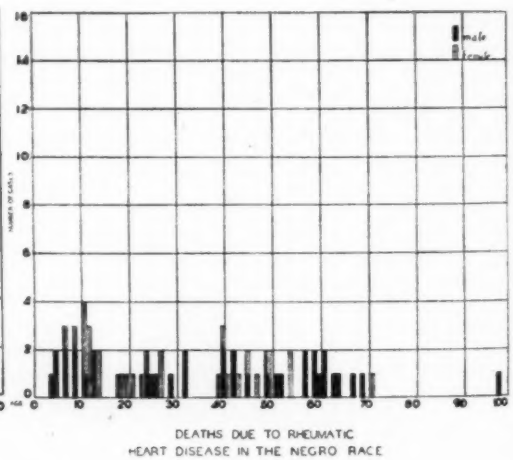

Fig. 8

(fig. 8). As will be seen in the graph there is a sharp rise in incidence of negro deaths in the adolescent period. A second rise occurs in the middle age group, at the age of 45 in the negro and at 60 in the white race.

There were 16 cases of subacute bac-

terial endocarditis, 11 of arteriosclerosis, five of acute bacterial endocarditis and five of hypertension in this entire group.

CONGENITAL

There were 23 deaths or 2. 2 per cent due to congenital heart lesions. These were subdivided into four white males, five white females, two negro males, and 12 negro females. The majority of deaths in this group were in infancy and the oldest was 16 years of age. This may explain the occurrence of only one case of subacute bacterial endocarditis.

TOXIC

There were 22 deaths (2.1 per cent) due to toxic heart disease, eight white males, five white females, five negro males, and four negro females. Of these 12 were due to diphtheria, four to typhoid fever, two to septicemia, two to pertussis, one to bronchopneumonia, and one to overdosage of digitalis.

BACTERIAL INFECTION

There were 14 deaths due to subacute bacterial endocarditis, six to acute bacterial endocarditis, one to acute postpneumonic suppurative myocarditis and pericarditis, and one to tuberculous pericarditis causing cardiac tamponade. There were 13 white patients, nine males and four females; and nine negro patients, six males and three females. None of these were found to show any evidence of pre-existing heart disease of any type.

ANEMIA

There were four white and three negro deaths due primarily to anemia, a total of seven or 0.7 per cent. The negro patients all had sickle cell anemia, while two of the white patients had pernicious anemia, one had sprue and one had severe secondary anemia due to chronic pulmonary hemorrhage.

AVITAMINOSIS

Two white males, one negro male and two negro females died from beri-beri heart disease.

HYPERTHYROIDISM

Three negro males and one negro female died of congestive heart failure secondary to thyrotoxicosis.

PULMONARY DISEASE

Four negro patients died from heart disease secondary to pulmonary disease. Two females and one male had Ayerza's disease ane one male had chronic cor pulmonale secondary to pulmonary tuberculosis.

OTHER TYPES

This group included a negro female and a negro male with amyloidosis of the heart and a negro male developed acute dilatation of the heart secondary to a transfusion given too rapidly.

UNKNOWN

One case of diffuse, non-suppurative, isolated myocarditis and one case of acute cardiac dilatation were included in this group. Both were negro females.

TRAUMA

There was one death in a negro female due to a stab wound severing the anterior descending coronary artery.

COMBINED

This group contained five cases showing two or more etiologic types of heart disease which all contributed to final death. Two white women, 23 and 20 years of age, had congenital and rheumatic heart disease complicated by bacterial endocarditis. One white woman, age 78, had a syphilitic saccular aneurysm of the aortic arch which terminated in a dissecting aneurysm extending into the common iliac vessels. A white man, age 76, had rheumatic and syphilitic valvular lesions and severe coronary arteriosclerosis. A negro man, aged 55 years, had rheumatic, hypertensive, arteriosclerotic heart disease with fenestration of the cusps of the aortic and pulmonary valves and an aneurysm of the right coronary artery 2.5 inches in diameter.

SUMMARY

A statistical survey of 1,045 cardiac deaths occurring in the Charity Hospital of Louisiana at New Orleans between the years of 1935-1940, inclusive, has been presented and analyzed according to age, race, sex, and etiology.

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DISCUSSION

Dr. W. A. Sodeman (New Orleans): The study carried out is important to every one of us. We are continually talking about and estimating the kinds of heart disease we see in south Louisiana and have not had reliable figures. As usually estimated, rheumatic fever amounts to 10 per cent of the total for Galveston and other centers, but the figures for our own area have never been satisfactorily analyzed. This study therefore is an important one.

There are some things that Dr. Holoubek did not say that I know he did because I have read his report in one of the medical journals. The incidence of these various forms of heart disease depends upon the case load, and upon the number of white and colored beds in the institution. If these factors are not taken into consideration the figures do not mean anything. Such factors I know have been taken into consideration in the present study.

There has been a lot of discussion about rheumatic heart disease in the South. Last month in one of our staff meetings it was stated that there is an increase in frequency of rheumatic fever at the present time, that ten or twelve years ago the incidence of rheumatic heart disease was very low and that it has increased remarkably since that time, especially in the last year or two. One of the men from the North said you have to educate the boys from the South about rheumatic heart disease. This should not be true of boys from Louisiana. I have been teaching in Tulane since 1932 and I do not know of any time when we have not been able to show the chronic stages of rheumatic heart disease. We have the major kinds that you see practically all of the time. We are having more of it now. Forty per cent of our cardiacs are not rheumatic as in New England but we have always had a sufficient amount of material as far as rheumatic heart disease is concerned, to teach its recognition. I went to medical school in the northern part of this country. I saw more than I see here; more on the wards there than here, but there has not been any dearth of material in New Orleans as far as rheumatic heart disease is concerned. Dr. Holoubek's figures are taken from 1935 to 1940 when some have said we have not had much rheumatic disease; you see we have. For the last two years I am sure the incidence of rheumatic fever would be increased in comparison.

Another thing that impresses me in the figures for rheumatic disease is that the incidence in white and colored is comparable with an edge for the colored race. Most of the colored which we have seen are Louisiana products, discounting the idea that our rheumatic patients come from the East or North. Most of the colored people are from Louisiana and most of them have not been out of the state. They got their rheumatic fever here; it started here and they started here. The disease in its course and incidence is entirely a product of this state. I wanted to take this opportunity to impress on you the importance of Dr. Holoubek's figures in connecting some of the remarks made in past years about rheumatic heart disease and its incidence here.

Dr. A. A. Herold (Shreveport): The excellent paper to which we have just listened dealt with heart disease itself. It would be interesting in closing if Dr. Holoubek would give a comparison, not as to whether rheumatic heart disease is more common in other climates but whether other discases such as scarlet fever, whooping cough and diphtheria are more common and therefore more heart disease attributable to that. If he would give us an idea as to comparative figures it would be interesting. It is not quite on the subject of his talk but would be of interest.

Dr. E. L. Wenk (Cloutierville): In Dr. Hunter's article of the current issue of the J.A.M.A., he stresses the absence of pain in myocardial infarction in the colored race. I have had several proved cases of myocardial infarction in colored persons in my private practice and pain has been marked in each case. I wish to ask Dr. Holoubek if he can give us any further information on this question.

Dr. J. P. Sanders (Shreveport): I would like to ask if there has been any change in the incidence or percentage of syphilitic heart disease in the colored race in the past few years. In Galveston, Texas, in 1926 the percentage of deaths in heart disease in the colored was higher than the percentage here. Obviously in the past years we have been putting on venereal disease campaigns and I wonder if this has had any effect on the result of heart disease.

Dr. Joe E. Holoubek (In closing): The autopsies performed at Charity Hospital are about three colored to about two white and that does not upset our statistics any; just about the same as inci-

dence, two-thirds of the patients who died were colored and one-third white.

We did not go over the clinical records to ascertain pain or anything like that, so I can not answer the question of pain in the negro. Many persons with coronary occlusions came in dead. If the diagnosis could not be made at autopsy then we went to the clinical record and reviewed it but practically all of the diagnoses were made in the autopsy room, so I am unable to answer the question.

Rheumatic heart disease does occur down here but rheumatic fever in its clinical form probably is not seen as frequently. In reviewing some of the percentages found in other places, New England, Chicago and elsewhere, in Chicago the incidence is 26.2 per cent hypertensive; 24.1 per cent arteriosclerotic; 9.7 per cent syphilitic and 29.2 per cent rheumatic. Further north in the New England states there is 29.2 per cent hypertensive and 39.5 per cent rheumatic, 25.7 per cent arteriosclerotic and 3.9 per cent syphilitic.

The incidence of syphilitic heart disease is, of course, much higher here and in Galveston; farther North the incidence is lower because the colored population is not very high. The majority of statistics in the North do not separate the white from the colored.

I do not have recent statistics as to whether the deaths from syphilitic heart disease are decreasing. You must remember all of these were deaths due to heart disease. If a man died from syphilitic heart disease he probably had primary lesions years before. I can not give information as to whether or not this has decreased; it is still prevalent.

REPAIR OF CHOLEDOCHUS DEFECT BY HEPATICODUODENOSTOMY OVER VITALLIUM TUBE

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The incidence of operative injury to the common bile duct has increased during the last decade. This is a direct result of the increased number of cholecystectomies which are now being performed for gallbladder disease. In former years, when cholecystostomy was the most popular biliary operative procedure, there were less reported instances of injury to the choledochus.

However, it is now generally accepted that cholecystectomy is the operation of choice in acute and chronic cholecystitis. Therefore, common duct injury has become a greater problem.

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Injury to the choledochus (actual severance, trauma resulting in stricture, or suture placed around the duct) practically always occurs in an attempt to perform simple cholecystectomy. There are three important reasons for this: (1) the failure of many surgeons to visualize clearly the common bile duct before clamping off the cystic duct and artery; (2) anatomic variations of the common, cystic and hepatic ducts and the hepatic and cystic vessels; (3) excessive traction on the fundus of the gallbladder by means of clamps (which produces angulation of the common duct).

Contrary to statements made by those who oppose frequent common duct exploration and drainage, injuries to the common duct occur more often during simple cholecystectomy than during choledochostomy. This indirectly proves the importance of visualizing adequately the choledochus when performing simple cholecystectomy.

There have been rare instances in which common duct injury has resulted during other operative procedures.[2] The most common of these is gastric resection for duodenal ulcer. It is easy to understand how the choledochus may become involved in adhesions from a long standing chronic duodenal ulcer and can possibly be injured in attempting to perform subtotal gastrectomy with actual removal of the ulcer. However, these cases are relatively uncommon as compared to instances of common duct injury during attempted cholecystectomy.

Recognition of choledochus injury at the time of the original operation is extremely important. If the surgeon is aware of the injury at this time, immediate repair is possible and is technically much less difficult than repair performed at secondary operation.

At the original operation, if the duct has been accidentally injured the severed ends of the duct are much easier to identify because of the absence of adhesions. Furthermore, the repair of the duct is less difficult because retraction and contraction of the cut ends of the duct have not yet taken place.

Maes[5] has recently stressed the importance of immediate recognition of common duct injury with repair of the defect at the original operation. He has reported a case of complete severance of the common duct during a difficult cholecystectomy. The injury was recognized immediately and the duct repaired. A rubber catheter was used because vitallium tube of the desired size was not available. The patient made an uneventful recovery and has remained well.

Various sizes of common duct vitallium tubes should be kept available in all hospitals where gallbladder surgery is done. Vitallium tubes are considered to be superior to the rubber catheter in the repair of common duct injuries.[2] There is a greater tendency for precipitation of bile salts in the lumen of the rubber tubes than in the vitallium tubes. Rubber acts strongly as a foreign body in the tissues, whereas vitallium is relatively inert.

Furthermore, the rubber tubing usually passes into the intestinal tract relatively early. The vitallium tubes on the other hand remain in place for a longer time (in all probability some vitallium tubes are never passed) and therefore there is less chance of subsequent stricture. The rubber catheter, however, has one advantage over the vitallium tube. The former is flexible and can be more readily bent to fit almost any type of common duct defect.

The exact type of operative procedure which is to be used in the repair of the choledochus defects (either stricture or severance of the duct or actual accidental removal of part of the duct) depends entirely on the size of the defect and the availability of the distal (lower) end of the duct.

If the defect is small and both ends of the duct can readily be isolated and brought together without tension, simple end to end anastomosis may be performed over a vitallium tube.[2] If it is impossible to approximate the two ends of the duct for direct suture, the tube may be held in place by infolding the peritoneal structures of the gastro-hepatic omentum. This may be reinforced with a fascial graft from the rectus sheath (Lord and Arthur).[4]

When a large defect is present, and particularly when the distal remnant of the duct is very small or cannot be found at all, some type of anastomosis between the common hepatic duct and the gastrointestinal tract must be performed. Various procedures have been advocated. The hepatic duct has been anastomosed successfully to the stomach, to the duodenum and to the jejunum. Hepaticogastrostomy is often unsuccessful because of the marked motility of the stomach. Hepaticoduodenostomy is technically easy if the duodenum is adequately mobilized. The anastomosis is usually secure because of the proximity of the second part of the duodenum to the porta hepatica and because of the fixation and lack of motility of the duodenum as compared to the stomach.[3, 6, 7]

Direct anastomosis of the hepatic duct to the jejunum is technically an easy procedure, but the postoperative incidence of cholangitis renders such a procedure prohibitive. Cholangitis occurs after hepaticoduodenostomy and hepaticogastrostomy, but the incidence does not near approach that of direct hepaticojejunostomy.

In recent years, Allen,[1] Cole[2] and others have advocated an anastomosis of the hepatic duct to an isolated loop of jejunum, using a modification of the Roux procedure.

By inverting folds of the intestinal wall into the isolated loop, a pseudo-valve arrangement is brought about and reflux of the intestinal contents into the biliary tree is cut down to a minimum and the incidence of cholangitis markedly reduced (Cole).[2]

This procedure is certainly a sound one from an anatomical and physiological standpoint and offers a new solution to this difficult problem.

CASE REPORT

Mrs. J. G., aged 42, was admitted to Touro Infirmary on April 12, 1944, for an elective cholecystectomy. For several years she had suffered from the usual symptoms of indigestion, flatulence, upper right quadrant pain and intolerance of fatty foods. She had never been jaundiced and there was no history of biliary colic, fever or chills.

On April 14 a cholecystectomy was performed.

The surgeon was of the opinion that the cystic duct and the artery had been cleanly clamped and ligated. There were no peri-cholecystic adhesions and by clamped traction on the fundus of the gallbladder it was possible to deliver the gallbladder well up into the incision. The procedure was not particularly difficult. In closing the gallbladder bed it was recorded on the operative note that there was a slight, persistent bile drainage.

The patient was seen by me in consultation on the fourth postoperative day. At that time, she was markedly jaundiced, the abdomen was distended and there was generalized rigidity and tenderness, slightly more marked in the right upper quadrant. The temperature was 103°, pulse 130, and the general condition of the patient was poor. The clinical picture was that of a generalized bile peritonitis.

On April 18, an emergency celiotomy was performed. Exploration of the biliary region revealed that the common hepatic duct had been severed close to the junction of the right and left hepatic ducts. The distal end of the common duct could not be located. Due to the patient's poor condition, no attempt was made to perform any type of anastomosis. A whistle-tipped catheter was placed into the hepatic duct and the subhepatic region was drained with several Penrose drains.

The patient's postoperative course was relatively uneventful. She immediately developed an external biliary fissure and the jaundice subsided. Her general condition improved rapidly.

On June 17, 1944, a hepaticogastrostomy was performed. This was done over a small vitallium tube and was successful for a period of four weeks. However, after a bout of severe abdominal pain the patient again developed an external biliary fistula through the old operative incision.

On August 22, 1944, celiotomy was again performed. The severed hepatic duct was located without difficulty. The duodenal arch was reflected medially and a small remnant of the distal common duct was found in the retroduodenal space. This stump of the common duct had a ligature still around it and had apparently been ligated by mistake for the cystic duct. The defect between the two severed ends of the duct was bridged with a relatively long, thin, vitallium tube. The peritoneum of the free margin of the gastro-hepatic omentum was folded over the tube by means of interrupted sutures of silk in order to hold the tube in place. This procedure was successful for only a period of five days, after which time the external biliary fistula again developed.

On October 16, 1944, celiotomy was again performed. An anastomosis was made between the hepatic duct and second part of the duodenum after the latter had been mobilized. The anastomosis was performed over a short, funnel-shaped vitallium tube. The funnel end of the tube was placed on the duodenal side and the anastomosis was performed with interrupted sutures of silk. This anastomosis has been successful and the patient has been free of jaundice and external biliary drainage. However, she has suffered three mild attacks of cholangitis which has subsided spontaneously in a period of three to four days. She is in excellent health and has gained approximately 23 pounds in weight.*

*This patient has recently returned because of severe recurrent attacks of cholangitis. There has been no jaundice or evidence of obstruction whatsoever, and it is believed that the passage of food material through the duodenum precipitates these attacks of cholangitis. In the near future, unless the attacks subside under conservative treatment, the patient will have a gastro-enterostomy performed with a surgical occlusion of the pylorus. Due to the fact that there is no evidence of obstruction of the hepatoduodenal anastomosis, it is not deemed necessary to perform the operation described by Allen, Cole and others.

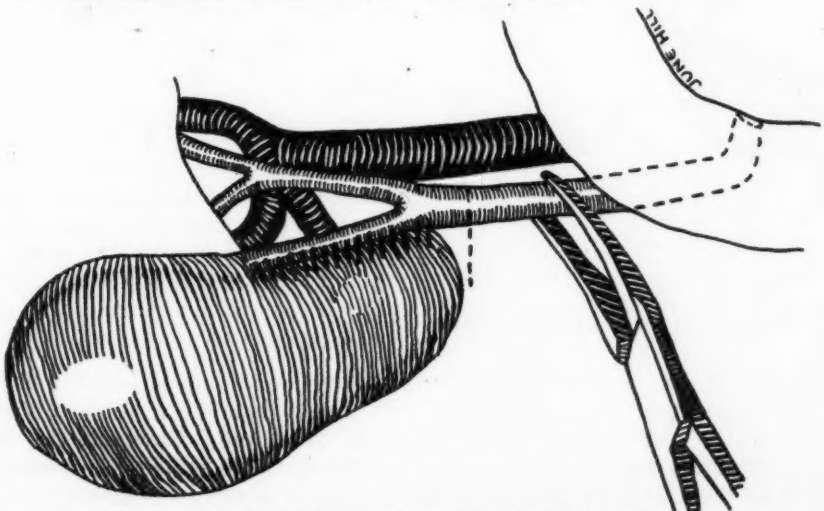

Fig. 1: Distended Hartmann's pouch adherent to cystic duct.

COMMENT

This case illustrates the complications resulting from accidental removal of a segment of the common bile duct in performing cholecystectomy. Furthermore, it shows the difficulties encountered when this error is not recognized at the primary operation. Persistent bile leakage into the subhepatic region during cholecystectomy is often indicative of hepatic or common duct injury and should always be carefully investigated from this standpoint.

There are a number of possible explanations of the choledochus injury in the case herein reported. In some instances, Hartmann's pouch is distended and adherent to the cystic and common ducts. The latter may be clamped by mistake for the former (fig. 1).

The cystic duct varies in length and occasionally runs parallel to the common-hepatic duct before joining the same to form the choledochus. If careful dissection is not carried out in such cases, either the choledochus or common-hepatic duct may be injured.

Excessive traction on the fundus of the gallbladder (fig. 2) may so angulate the

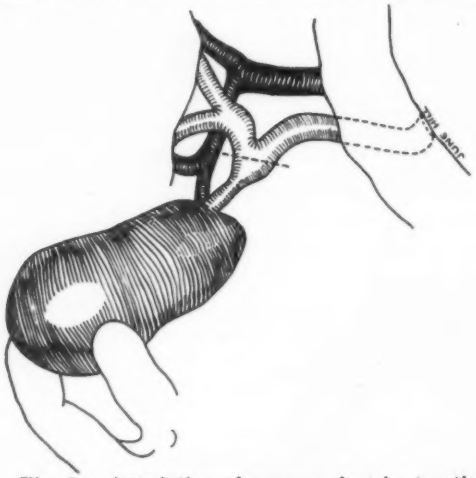

Fig. 2: Angulation of common duct by traction on fundus.

choledochus that it may be clamped by mistake for the cystic duct.

Bridging of the choledochus defect by means of a vitallium tube connecting the severed ends of the duct is the procedure of choice. This procedure more closely restores the normal anatomy. However, such a procedure is difficult if the defect is large, as in this particular case.

Hepatogastrostomy is a relatively simple technical procedure. However, the marked mobility of the stomach often will cause such an anastomosis to leak and eventually break down completely. This, I believe, happened in the particular case which is herein recorded.

An anastomosis of the hepatic duct to the duodenum was successful in this case, (fig. 3). Such a procedure is preferred by some

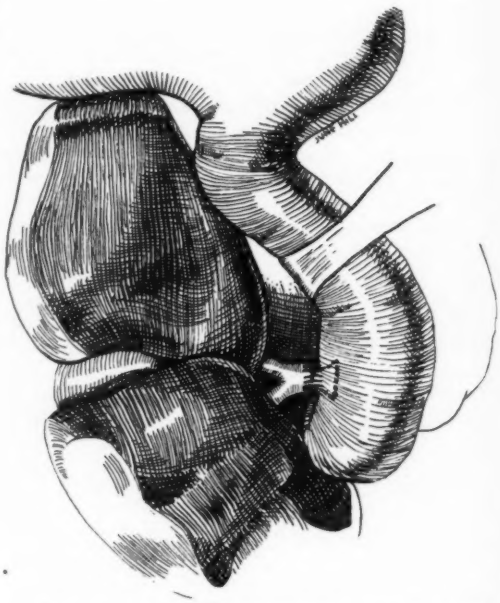

Fig. 3: Hepaticoduodenostomy over funnel-shaped vitallium tube.

surgeons (Sanders,[6] Dragstedt[3] because the bile normally enters the duodenum and the relative immobility of the latter renders stability to the anastomosis. However, cholongitis will sometimes occur and may cause postoperative difficulty.

Direct anastomosis between the hepatic duct and jejunum is not a difficult technical procedure. However, the high incidence of severe cholangitis renders such a procedure practically useless.

In recent years, Allen[2] and others have advocated the anastomosis of the hepatic and common bile duct to an isolated loop of jejunum. A modified type of Roux procedure is performed in order to isolate a loop of jejunum. The latter is then anastomosed to the biliary tree. Pseudo-valves in this loop of jejunum are then formed by infolding the wall of the bowel and the biliary tree is well protected against ascending infection from the gastrointestinal tract.[2] The incidence of cholangitis is practically nil after such a procedure.

It is entirely possible that this last operation may become necessary in the case herein reported, if the attacks of cholangitis become more severe and more frequent and if there is any evidence of inhibited hepatic function or obstruction of the hepaticodudenal anastomosis.

SUMMARY

1. An actual increase in the number of operative common duct injuries has occurred in recent years because of the increased number of cholecystectomies now being performed.

2. The high incidence of anatomic variations in the bile ducts and vessels predisposes some patients to common duct injury.

3. It is imperative to recognize a common duct injury at the primary operation in order to perform immediate repair. Greater technical difficulty is encountered at secondary operation because of extensive adhesions, presence of biliary fistula and retraction and contraction of the ends of severed choledochus.

4. Reconstruction of the common bile duct over a vitallium tube is the treatment of choice with end to end anastomosis.

5. Vitallium tubes are superior to rubber tubing because of the lesser degree of tissue reaction and deposits of bile salts therein. The vitallium remains in place longer and may prevent subsequent stricture formation.

6. When a large defect in the common bile duct is present, it is usually necessary to anastomose the common bile duct to some part of the gastrointestinal tract.

7. The various advantages and disadvantages of hepatogastrostomy, hepatoduodenostomy and hepatojejunostomy are discussed.

8. Recent reports indicate that an anastomosis of an isolated loop of jejunum to the choledochus is the most successful operative procedure in such cases.

9. A case is reported herein, in which a large part of the common bile duct was removed accidentally in performing cholecystectomy. Subsequent reconstruction of the common duct and a hepatogastrostomy was unsuccessful. Hepatoduodenostomy has been performed. The latter has been successful, but several mild episodes of cholangitis have occurred.

10. It is possible that re-anastomosis of the common duct to an isolated loop of jejunum may be necessary at a latter date in this particular case because of the complicating cholangitis.

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FEVER OF UNDETERMINED ORIGIN

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NEW ORLEANS

Febrile illnesses are not uncommon in the South; even with the best of diagnostic facilities, many are undiagnosed. It is not at all unusual to see a patient with febrile symptoms (headache, malaise, vague somatic distress, nervousness, exhaustion, pal-

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pitations and weight loss), but no physical signs of a specific entity. Accordingly, the diagnosis "fever of undetermined origin" has become accepted generally. Needless to state, such a "diagnosis" is warranted only if the cause for the pyrexia is not evident after prolonged and thorough search for the cause.

In the tropics, obscure fevers are frequent.[7] In the temperate zones these febrile states are encountered more than is generally realized. Sodeman[25] observes that what is the common cause of fever in one locality may be only an occasional etiologic factor in another, more remote site. Thus one would not necessarily search for the same causes of fever in New England as in the deep South. In similar fashion, in ascertaining the cause of a febrile episode in a man returned from overseas military duty, one would like to know what diseases prevail where the man served.

Within recent years rheumatic fever has become often suspected as the cause of an obscure febrile state. We know now that in a large proportion of cases of rheumatic fever, the classical signs are lacking. In Boston, as well as in New Orleans, studies have indicated that the majority of proved instances of rheumatic carditis are not preceded by arthritis, skin manifestations and chorea. Is it possible that many cases of "fever of undetermined origin" are due to an undiagnosed rheumatic infection?

The current interest in psychosomatic matters focuses attention on the question: Can fever be caused solely by a neurogenic or psychogenic process? Evidence to this end has been furnished by Reimann[24] and by Rappaport.[29] The latter observer reports a series of cases in which there was disproportion between the oral and the rectal temperatures. The oral temperatures rose above normal, whereas the rectal level constantly remained normal. This condition, which he calls "essential oral hyperthermia", was unaccompanied by any evidences of organic disease; in each instance, he attributes this thermal paradox to instability of the heat regulating mechanism in psycholabile individuals.

The purpose of our study was to determine the eventual outcome of a series of cases whose hospital diagnosis was "fever of undetermined origin." Similar studies (Alt and Barker,[2] Hamman and Wainright,[10] Keefer,[13] and Kintner and Rowntree[14]) have attested to the desirability of reviewing such cases. Since all our patients had their daily temperature variations recorded by oral methods, we have adhered to the standard that anything over 99.8° F. constitutes fever.

CAUSES OF FEVER

The heat controlling and heat regulating centers are believed to be located in the caudal part of the lateral hypothalamus and are apparently influenced in two ways—reflexly from the skin, and by the temperature of the blood flowing through them. The centers control body temperature by the transmission of sympathetic impulses to the cutaneous vessels, sweat glands, and pilomotor muscles. The thyroid and adrenal glands play their roles in the regulation of body temperature through the calorigenic effects of their secretions. Since the body temperature represents the balance between heat production and heat loss, a disturbance in this balance will be followed by a temperature change.

Carr[5] cites Leschke's classification of fever as due to:

(a) Physical changes of the surroundings—exhaustion or inadequacy of the heat dissipating mechanisms, as in heat stroke.

(b) Injuries to nervous centers—especially lesions of the following regions: third ventricle, internal capsule, medulla, or upper part of the spinal cord.

(c) Drugs and other chemical substances—dinitrophenol, large doses of adrenalin or thyroxin.

(d) Substances which arise from the destruction of body cells with liberation of toxins.

(e) Substances which arise from pathogenic microorganisms in an infected body.

If the cause of fever is not found by complete general and regional examination, it is well to consult the table of possible causes

as suggested by Albrecht,[1] Harrell[11] and Keefer.[13]

I. SPECIFIC INFECTIONS
- A. Septicemia (anaerobic streptococci, brucella, neisseria, eberthella typhosa, *Hemophilus influenzae*, pasteurella tularense, staphylococci).
- B. Secondary syphilis
- C. Malaria
- D. Generalized infections
  - 1. Rickettsial group
  - 2. Rheumatic fever
  - 3. Relapsing fever
  - 4. Rat bite fever
  - 5. Trichinosis
  - 6. Lupus erythematosus
  - 7. Periarteritis nodosa
  - 8. Infectious mononucleosis
  - 9. Tuberculosis
- E. Localized infections
  - 1. Renal and perirenal
  - 2. Hepatic (amebiasis)
  - 3. Subdiaphragmatic abscess
  - 4. Bronchiectasis
  - 5. Osteomyelitis
  - 6. Cholecystitis
  - 7. Thrombophlebitis
  - 8. Prostatitis
  - 9. Sinusitis
  - 10. Dental infections

II. TUMORS AND MALIGNANCIES
- A. Generalized carcinomatosis, especially with liver metastases

III. DISEASES OF THE BLOOD FORMING ORGANS
- A. Lymphomas
- B. Leukemias
- C. Pernicious anemia

IV. DISORDERS OF THE HEAT REGULATORY MECHANISM: METABOLIC DISORDERS
- A. Hyperthyroidism

V. MISCELLANEOUS
- A. Drug reactions
- B. "Psychogenic" fever
- C. Malingering

PROCEDURES SUGGESTED FOR STUDY OF OBSCURE PYREXIA

Blood — Complete blood picture, sternal marrow smears, studies for malaria, blood chemistry and Wassermann, repeated blood cultures (special media and anerobic conditions) agglutination studies, and sedimentation rates.

G. I. Tract—Gastrointestinal x-ray series, barium enema, proctosigmoidoscopy, stool studies for ova, parasites, and bacterial pathogens. Hepatic function tests, and cholecystograms.

Cardiorespiratory—Sputum studies, x-ray of the chest, electrocardiogram, bronchoscopy.

Genitourinary Tract—Urinalyses, prostatic secretion studies, cystoscopy and pyelography.

Other Tests—Lumbar puncture, x-ray studies of sinuses and teeth, basal metabolic rate determinations, biopsies (lymph gland, liver, skeletal muscle, skin, cervix).

CASE SERIES

Our cases were selected from 66,565 admissions to the Touro Infirmary between January 1, 1940, and December 31, 1944. All records with the primary diagnosis of "Fever of Undetermined Origin" were studied. These numbered 189, and from them there were chosen 45 records of patients 13 years of age or older with obscure pyrexia lasting at least 10 days in which no definite diagnosis had been made after thorough studies during hospitalization.

Follow-up letters were sent to the physicians who had treated these patients and subsequent data were obtained on 36 of the 45.

FOLLOW-UP OF 36 CASES OF FEVER OF UNDETERMINED ORIGIN

In only four cases was a definite diagnosis ever established. Of these three have died, and one was living and well when last seen three years after hospitalization for obscure pyrexia. These cases are reported in abstract below.

In 32 cases no definite diagnosis was ever established. Of these, 26 are living and well at the time of writing this paper. Four were living and well when last seen one and a half to two and a half years after admission for fever of undetermined origin. One is living and still ill (case abstract below). One is living and has carcinoma which is not considered the cause of his previous pyrexia (case abstract below).

ILLUSTRATIVE CASE REPORTS

In addition to the abstracts of six cases mentioned above we are reporting a case summary of a patient with fever seen here in 1945 in whom the ultimate diagnosis was Hodgkin's disease, and two cases from the senior author's practice. One illustrates recovery from recurrent bouts of fever without any diagnosis ever being reached. The

second ultimately proved to be probably one of subacute bacterial endocarditis.

Specific infection: endocarditis diagnosed at autopsy.

A 49-year old white male was admitted January 29, 1941, with a history of fever of one month's duration. Past history revealed only "typhoid fever" at the age of 23. Examination showed pyorrhea and a loud, rasping mitral systolic murmur. During six weeks of hospitalization he had intermittent type of fever with almost daily elevations to 101 or 102° (F.). Five blood cultures, repeated malaria smears, sputum studies, agglutination (typhoid-typhus-melitensis groups) reactions, complete blood pictures, urinalyses, and serology, all were non-contributory. X-ray studies demonstrated normal sinuses; the chest showed increased pulmonary vascular markings, a slightly diffusely dilated aorta with calcium deposition in the arch, and the residuum of an old minimal (arrested) tuberculous lesion at the level of the right first interspace. Nothing suggesting active disease was seen in the chest plate. An electrocardiogram was abnormal (PR 0.12 seconds, QRS 0.06 seconds, sinus tachycardia rate 180 per minute, T-1-2-3 low or flat, S-1-2-3 slurred). Clinical impressions were questionably active rheumatic endocarditis or subacute bacterial endocarditis. Treatment with sulfanilamide and pyramidon gave no results other than temporary afebrile periods. On March 16, 1941 he was transferred to another hospital, where he expired in July, 1941. Autopsy showed large verrucous vegetations on the aortic and mitral valves; cultures from these vegetations were negative.

A 75-year old white male was admitted April 20, 1942 complaining of fever for 10 days, earache, sore throat, and cough. Cholecystotomy in 1939 represented the only previous operation. Examination showed no significant findings. During three weeks of hospitalization there was an irregular, intermittent fever exceeding 100° (F.) at times. The total white cell count was 14,000 (84 per cent neutrophiles, 3 per cent eosinophiles, 11 per cent small lymphocytes, 2 per cent monocytes); urinalysis, sputa, serology, malaria smear, blood culture, agglutinations (typhoid-typhus-melitensis-tularemia groups) all were negative. An electrocardiogram was within normal limits. Three x-ray studies of the chest at weekly intervals revealed arteriosclerosis of the aorta, osteoarthritis of the spine, increased right lung markings, and some enlargement of the right hilus; no gross infiltration was seen. The radiologist could not differentiate between interstitial pneumonitis or pulmonary congestion, and advised bronchoscopic study, which the patient refused. ENT consultant reported ears, nose, sinuses and throat as negative. Tentative diagnosis was carcinoma of the lung. No further studies were made, the patient was given a course of sulfathiazol, and discharged home, still having low grade fever on May 11, 1942. He died a few months later; autopsy revealed bronchiogenic carcinoma.

Diseases of the blood forming organs: Hodgkin's disease diagnosed by biopsy.

A 55-year old white female was seen in Medical Clinic in May, 1945, complaining of chills, fever, and profuse sweating of six months' duration. She was admitted to the ward June 6, 1945, where further studies revealed only a past history of umbilical herniorraphies in 1933 and 1934; complete regional physical examination was negative. During this first study an intermittent fever of 101 to 103° (F.) was noted on four consecutive days. The total white blood count was 15,800 (70 per cent neutrophiles, no eosinophiles, 30 per cent small lymphocytes); four malaria smears, agglutination (typhoid-typhus-melitensis-heterophile groups) reactions, blood NPN and sugar, cephalin flocculation, icterus index, stool culture, and blood culture, were all noncontributory. Sedimentation rate was markedly increased—110 mm. per hour. She was discharged afebrile June 15, 1945, the etiology of her fever still uncertain.

She was readmitted August 1, 1945 with the same complaints of fever and weakness. Physical examination again was negative. During this second period of 27 days' study a typical Pel-Ebstein fever curve was noted; a recurrent, remittent pyrexia with range between 100 and 103° (F.) for four days, followed by an afebrile period of five days, and then a five-day cycle with a temperature range between 100 and 103° (F.). Repeated search for evidence of lymphadenopathy, splenomegaly, or hepatomegaly was unsuccessful. The leukocytes numbered 12,800 per cubic millimeter (differential as on first admission). Sedimentation rate was 115 mm. per hour. Repeated malaria smears, blood cultures, stool cultures and examinations for ova and parasites, and a brucellergin skin test, all yielded negative results. The urologic consultant found a urethral stricture and a low grade cystitis, which were treated successfully and were not considered to be the cause of the fever. A KUB and a chest plate were negative. Emetine was administered for a possible hidden amebic hepatitis; penicillin was also given. No effect on the fever was noted. The impression was possible Hodgkin's disease. On August 28, 1945 she was discharged to the medical clinic. There a trial of atabrine and quinine was administered but without effect on the febrile recurrences.

In about December, 1945, in another city, the patient first became aware of a mass in her left axilla. She was admitted to another clinic, where the lesion was excised. The report of the pathologist was forwarded to us, and confirmed Hodgkin's disease.

The patient returned to the Touro Out-Patient

Clinic in late December, 1945, and has subsequently received a series of deep x-ray treatments for a group of enlarged glands in the left supraclavicular region. At present (March, 1946) she continues to have recurrent bouts of fever, has a negative chest plate, and no other signs of lymphadenopathy, splenomegaly, nor hepatomegaly. There has been no weight loss.

Disorders of the heat regulatory mechanism: hyperthyroidism.

A 29 year old white female was admitted May 20, 1941 complaining of low grade fever, chills, weakness, loss of appetite, and weight loss, of four to five months' duration. Past history was noncontributory and examination (including pelvic) revealed only a congenital erosion of the cervix. During the three weeks of study her temperature revealed a low grade, irregular fever of between 99 and 100° (F.), white blood count was 10,500 with 76 per cent neutrophiles and 24 per cent small lymphocytes. Urinalysis, serology, agglutinations (typhoid-typhus-melitensis-tularemia groups) stool and blood cultures, all added no information. A BMR was minus 6 per cent. X-rays of the chest and gastrointestsinal series were negative for pathology. She was given one blood transfusion (red blood cells 4,200,000 with 80 per cent hemoglobin on admission) and was discharged on June 13, 1941 still febrile, following courses of sulfathiazol and sulfapyridine.

One year later definite findings of thyrotoxicosis were found by her physician, a successful thyroidectomy was performed, and she was in good health when last seen in 1944.

Miscellaneous causes: cirrhosis of the liver. Diagnosis established on subsequent admission, with fatal termination.

A 57 year old white female was admitted on August 4, 1941 complaining of low grade fever, cough, and nausea for four months following a bout of "influenza." There was a past history of hematemesis in 1940 attributed to a possible duodenal ulcer. Examination showed only poor oral hygiene, hypertension, and a soft systolic apical murmur. During the four days of study there was a regular daily temperature rise to 100° (F.). The liver and spleen were palpable at no time, and no skin lesions were noted. Gastric analysis showed no free hydrochloric acid and nine degrees of total acidity. Blood NPN, sugar, CBC, melitensis agglutination and skin test, and serology were all negative. X-ray of the chest demonstrated a prominent left ventricle and a dilated thoracic aorta. A gastrointestinal series was negative. No treatment was given, no clinical impression was formed, and she was discharged still febrile on August 8, 1941.

She was readmitted on October 11, 1941 with a typical clinical picture of terminal liver failure and expired on October 27, 1941. At autopsy a Laennec's cirrhosis of the liver was found.

Recurrent fever more than three years: Definite diagnosis has never been established.

A 13 year old white female was admitted on July 29, 1942 complaining of fever of 10 days' duration. There was a past history of recurrent "pyelitis" of "12 years' duration." The patient attributed the present bout of fever to an injection of typhoid vaccine received the day the pyrexia began. Examination revealed an acutely ill, somewhat lethargic girl; the nasopharynx was injected, the spleen just palpable, and there were a few moist rales at the left lung base. During the five weeks of study a septic and remittent type of fever was noted almost daily, ranging from 100 to 103° (and at times to 104°) (F.). Repeated CBC's showed a WBC range usually under 10,000; the lowest was 4,800 and the highest 12,500. The differential count typically was: 72 per cent neutrophiles, 3 per cent eosinophiles, 22 per cent small lymphocytes, and 3 per cent monocytes. Urinalysis showed a few casts and sulfathiazole crystals on admissions, but repeated specimens (including catheter specimens) were negative. Blood agglutination (typhoid-typhus-melitensis-dysentery groups) studies were repeatedly negative, as were stool and blood cultures. Spinal fluid pressure on admission was 40 cm. water; a later one, 155 cm. water, but chemical, serologic, and bacteriological studies of these specimens gave no positive findings. X-ray studies of the chest and abdomen were negative. Electrocardiograms showed tracings within normal limits (P-R intervals 0.15 and 0.13, QRS intervals 0.07, right axis deviation-slight). An EENT consultant found no abnormalities. During the period of hospitalization, the lethargy cleared. A faint "butterfly rash" was noted by several observers over the bridge of the nose. There was no response to courses of quinine and of sulfadiazine. The febrile course persisted; on the day she left the hospital (August 26, 1942) a faint pericardial friction rub was first noted. Clinical impressions were: possible disseminated lupus erythematosus, miliary tuberculosis, or virus encephalitis.

Her physicians report (March, 1946) that she is still alive, has had a chronic febrile course over subsequent years, has been treated by several other physicians, and that no definite diagnosis has ever been established.

Presumptive diagnosis: subacute bacterial endocarditis—two cases.

Mrs. E. F., 41 year old white widow, reported for examination August 25, 1945 because of fever of 18 days' duration. This fever tended to rise as high a 103°, usually in the afternoon but at times in the morning, and was accompanied by profuse perspiration, malaise and exhaustion. As a rule, it would subside after three or four hours at the end of which time the patient would feel well enough to undertake her customary household duties. The single physical finding of note in the

afebrile phase was a systolic apical murmur. Since one of her brothers had died of subacute bacterial endocarditis, the suspicion of this illness was constantly entertained. Repeated blood cultures were negative by the aerobic and the anaerobic methods. On at least six occasions, attempts were unsuccessfully made to demonstrate agglutinins against the various strains of brucella, typhosus, paratyphosus, tularense and B. Proteus OX 19. Samples of blood were submitted to various laboratories, including that of the National Institute of Health, for these determinations; none was ever recorded as positive. Blood was injected into guinea pigs; no pathologic process was demonstrated. Examinations for such rare conditions as rat bite fever were conducted with negative findings. Thick and thin smears for malaria plasmodia were negative. X-ray examinations of the chest, the teeth, accessory nasal sinuses, gastrointestinal tract were all within normal limits. No blood dyscrasia was demonstrable. Dental, urological and gynecological examinations were negative. Her fever continued in this fashion for about four months until one physician empirically administered 3,000,000 units of penicillin following which the fever and febrile symptoms disappeared and have not recurred to date. It is questionable whether this should be recorded as an instance of fever of still undetermined origin or whether one should accept it as a case of subacute bacterial endocarditis with no demonstrable bacteremia or skin manifestations, yielding to a relatively small amount of penicillin.

A. R. was a single, 28 year old white girl when first seen February 15, 1945. At this time she complained of painful and occasionally swollen ankles and intermittent fever. There was a history of rheumatic fever at the age of 12 years with incapacitation for one year. The details of this illness were unfortunately not available. She was pale, had a hypochromic anemia and there was a loud systolic and presystolic apical murmur. There was no x-ray evidence of pulmonary disease or of cardiac enlargement. Despite repeated attempts at blood culture, none was reported positive. The fever subsided after several weeks, the diagnosis "fever of undetermined origin" being made. Her symptoms did not respond to bed rest, large amounts of iron, and a high calorie, high vitamin regimen. She left the city for a period of two months' rest in the country and returned September, 1945, feeling much better and reporting "only occasional" fever. Again attempts were made to find the cause of the fever without success; she did not report again until September 15, 1945 at which time the fever had become steadily 103°. She was admitted to Touro Infirmary where a most extensive investigation was conducted without results. She awoke one morning to find some petechiae on the abdomen and on the hands and legs. Penicillin was begun in large amounts, and she received a total of 12,000,000 units in the suceeding two months. The fever receded somewhat and she was temporarily improved by repeated blood transfusions. At her own request she was discharged December 1, 1945. She was readmitted December 23, 1945 because she had awakened to find herself speechless and unable to move her right lower extremity. There were several new petechiae and the spleen was not palpable. The diagnosis of subacute bacterial endocarditis was made despite the continued negative blood culture. Sulfadiazine was given by vein and penicillin (total 18,000,000 units) by various routes. She soon made a speedy recovery and was allowed to go home February 28, 1946. She was afebrile for three months until following a dental extraction, her fever recurred and has persisted to date. There has been marked mental deterioration since this time and her general status closely approaches that of the senile person who has suffered a cerebral accident.

Undiagnosed fever: unrelated carcinoma developing eventually.

A 68 year old white male was admitted February 7, 1940 complaining of fever for about two and a half weeks, slight cough, some vague chest discomfort, anorexia, constipation, and mild abdominal fullness. There was a past history of typhoid fever at the age of seven years, tuberculosis at 28 years, and of known prostatic enlargement. Physical examination gave no remarkable findings, other than moderate prostatic enlargement. During the 10 days of hospitalization an irregular, intermittent type of fever was noted with rises of 100 to 101° (F.) almost daily. He had slight normocytic anemia, white blood cells of 25,000 and later 10,500 with 91 per cent and 74 per cent neutrophiles (respectively), and negative smear for malaria. Blood culture, serology, agglutinations (typhoid-melitensis groups), blood NPN and sugar were noncontributory. The genitourinary consultant found grade 3 benign prostatic hypertrophy, but after study of the urine and KUB could find nothing to account for the fever. X-ray of the chest showed no evidence of any active pulmonary disease; but there was hypertrophic osteoarthritis of the spine. Dental films revealed only alveolar resorption. After a short course of prontylin, the patient was discharged still febrile on February 17, 1940.

In April, 1943, he began to notice colicky adominal pain and melena. A barium enema revealed a carcinoma of the middle third of the sigmoid in September, 1943. He was readmitted on December 5, 1943, and two days later an end-to-end anastomosis with resection was performed. The pathologist reported adenocarcinoma with metastases to the pericolic tissues. There had been no fever since soon after leaving the hospital in 1940.

At the present time he is living and comfortable. A barium enema in January, 1946, showed diverticulitis and irritability of the colon just proximal

to the site of anastomosis, but no evidence of recurrence of the carcinoma.

DISCUSSION

The purpose of this study was to follow a series of patients whose febrile courses had not been satisfactorily diagnosed. It was appreciated here, as elsewhere in the domain of clinical medicine, that time is an essential factor in the diagnosis. If the patient is studied often enough and long enough, the diagnosis would become evident in most of them. In some instances, a diagnosis was established only at postmortem.

There still remained a group whose diagnosis was never satisfactory. What caused their "fever of undetermined origin"? The fact that 32 of the original group went without adequate diagnosis is impressive. Some of them had fever as long as two years without diagnosis of the cause. Several questions arise in our minds: How long can a patient continue to have fever without developing as an "interesting" phenomenon localizing diagnostic signs? How long can fever continue without curtailing a patient's physical abilities? How often do such patients spontaneously "burn out" their fever and cease to be patients? Now that powerful antibiotics are available, these questions are of more than academic interest. Are we justified in administering penicillin or streptomycin empirically to such patients, realizing that there is some risk in the development of a "penicillin-fast" state? Lastly the question arises: Is it possible that at least some of these instances are due to an unrecognized virus disease which eventually runs a self-limited course?

SUMMARY

1. Fever of undetermined origin has been briefly discussed with consideration of causes of fever, some of the more common disease entities ultimately diagnosed, and the procedure used in studying such cases.

2. Follow-up studies have been presented of 36 cases of obscure pyrexia.

3. Typical case abstracts have been given of undiagnosed, ultimately diagnosed and self-limited cases.

We wish to express our appreciation to members of the staff of The Touro Infirmary for allowing us to use their private case reports.

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CATARACT SURGERY AS DONE IN INDIA TODAY

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During the past winter I had the good fortune of being able to do eye surgery in India, and due to the transportation difficulties I was the only American over there doing eye surgery. This afforded me the unusual opportunity of working with the three leading cataract surgeons of India, Drs. Holland, McPhail and Mathra Das. These three men do more surgery than all the other eye men in India combined. I spent about six weeks with each of these men and performed over one thousand cataract extractions under their supervision. In addition, I observed over two thousand cataract extractions performed by these and other leading surgeons. I visited eye hospitals and talked to surgeons in Egypt, Arabia and Ceylon as well as in the larger towns and cities of India, and in all my travels I did not find two men using the same technic.

Few patients are seen before the day of operation, and then only a few hours before operation. No blood pressure is taken, no smear is made, and there is no chest or urine examination. The eye is inspected and if there is no apparent pus in the sac and the trachoma, which is present in approximately 97 per cent, is not too acute, and there is apparent glaucoma, (Sir Henry Holland takes tension after the patient is on the operating table), the eyes are operated on. If the eyes are glaucomatous an iridectomy is done, followed by removal of the cataract in three to ten days, depending on the operator. The majority of patients, however, are not operated upon if they can count fingers at twelve inches. Ninety-five per cent of these people are unable to read or write. Their chief requirement is to be able to distinguish a cow from a goat and to be able to walk about some. Of those operated upon, less than 10 per cent will get a plus ten sphere with no reading segment after operation. Therefore, if they can count fingers before operation, they are not satisfied with the operative results.

The patient is put on the table wearing the same dirty clothes in which he slept the night before. If a gown is used by the operator, it is for the protection of his clothes. No sterile piece or eye sheet of any kind is used. The eye is douched by an assistant with 1/2000 perchloride of mercury solution. The surgeon scrubs his hands with soap and water before starting the operations and not again unless he has to handle something other than the patient's eyes. He passes from one to the other but he uses sterile instruments for each eye.

Hospital beds, as we know them, are never seen except in a few hospitals in the largest cities where their total number of cataract extractions for twelve months is only a few hundred. A few fortunate patients get charpoys (rope cots) to sleep on but the average patient has some rice straw on the floor of a warehouse or on the ground under the trees. I have seen over 400 patients lined up in long rows on straw beds in a large warehouse. The patient is told to lie quietly for 24 hours but as the only nurse is a member of the family or a friend, he usually does more or less as he pleases in regard to nature's demands. The patient is allowed to go home about the tenth day. Surprising as it may seem under these conditions, infection (purulent endophthalmitis) occurs in only about one per cent of these cases.

As there is no standardized technic in either the capsulotomy or intracapsular method, I shall describe briefly the technic employed by each of the three surgeons with whom I worked, and also the different methods employed by other men with whom I visited.

Sir Henry Holland, who needs no introduction, has performed approximately 100,000 cataract operations in his hospital. About half of his operations are intracapsular; the other half capsulotomy. His criteria for the intracapsular method is an uncomplicated cataract in a calm patient over 50 years of age, not plethoric, without a bulging or "ox eye" and with the tension

(McLean) not over 30. If the tension is slightly raised, he does a posterior sclerotomy. He uses 4 per cent cocain and adrenalin for anesthesia, but if the patient is very nervous he performs the operation under general anesthesia, using chloroform.

In performing the intracapsular operation, Sir Henry Holland makes the incision entirely in the cornea from nine to three with no flap or bridge. He ruptures the capsule with the knife, often finishing the incision, and then does a broad iridectomy. An assistant keeps the eye fixed with forceps while the iridectomy is being done.*

In the capsulotomy method he uses the squint hood to express the lens while the spoon makes backward pressure on the scleral side of the incision, thereby causing a slight gap in the wound. On all patients he uses the O'Brien nerve block and also a suture in the upper lid which is taped to the cheek upon completion of the operation for the purpose of keeping the eye closed until the block has worked off. He operates on both eyes if indicated. The eye is dressed on the second day.

Dr. McPhail has performed some 50,000 cataract extractions in his hospital. He employs the capsulotomy operation exclusively, using cocain crystals for anesthesia. After the puncture is made, he ruptures the capsule with the point of the knife, then does the counterpuncture, and finishes the incision with a narrow conjunctival bridge about 4 mm. wide, slightly nasally, and then does a broad iridectomy by inserting forceps on the temporal side close to the bridge. He holds the speculum with the left hand and expresses the lens with a curved, glass lens expressor. The cornea is then massaged superior to the limbus with the lens expressor to remove any loose cortex. If the patient is very nervous he removes the speculum and delivers the lens by the same maneuvers, using the thumb on the brow and the middle finger on the lower lid to keep the eye open. Dr. McPhail does not have an assistant while performing this operation. The patient walks from the operation room and his eye is dressed on the third day.

Dr. Mathra Das, the Henry Ford of cataract surgery, has performed more cataract extractions than any man who has ever lived, and probably more than any other man ever will. Up to January 1, 1946 he had done 188,663 cataract extractions, and since that date he has operated upon an additional 2,000 or 3,000. His record for one day is 707 extractions. He uses four tables and his usual speed is one a minute if there are no interruptions. He holds "blind camps" all over India and Burma and has from 200 to 2,200 cataract operations in each camp. The last day I spent with him before I came home, we did 237 cataract extractions in 270 minutes. I did about one-third of the operations.

Dr. Mathra Das uses intracapsular method in all patients over 30 years of age unless patients have "ox eyes" or are very unruly. He uses 3 per cent butyn for anesthesia. If mild trachoma is present he paints the lids with 12 per cent silver nitrate and then operates with surprisingly good results. He makes the incision entirely in the cornea, finishing the incision about half way between the limbus and pupillary margin. This leaves a corneal scar but the average patient does not complain about it. He claims that lens delivery is easier with this type of incision.

In performing an iridectomy, Dr. Mathra Das uses curved iris scissors without the eye being fixed. He removes the speculum and an assistant takes care of the lids. The upper lid is held away from the eye by a squint hook while the operator's left thumb pulls back on the brow. At the same time the left index finger steadies the hook which can be moved by him in any direction for better exposure. The lens is expressed with the point of another squint hook, the pressure being made well below the limbus upward and backward so that the lens is tumbled with the lower edge presenting.

*Formerly Sir Henry Holland advocated performing this operation without having the eye fixed. However, he has changed his technic and now prefers to keep the eye fixed during operation.

If the capsule ruptures in this position, it is easily removed with forceps. The point of the hook follows the lens up, tucking the cornea under the lens, and if the lens sticks in the incision, the hook is pushed behind and the lens is lifted out. The patient's eye is dressed on the seventh day.

COMPLICATIONS AT OPERATIONS

Other than expulsive hemorrhage, accidents at the time of operation are directly proportional to the surgeon's ability. Vitreous loss is the most frequent complication in either method, and is about the same in each, 3 per cent. The loss of a few drops of vitreous does not seem to influence the outcome. All of these doctors claim that it does no harm.

Prolapse of the iris is a serious complication which occurs more frequently in the intracapsular than in the capsulotomy method, 3 and 1.5 per cent respectively. Some surgeons excise the prolapse at first dressing while others wait until the eye is quiet before doing anything. However, the tendency now is to wait about six weeks until the eye is quiet. The majority of these men do an iridectomy but there are no figures available to substantiate their claim of less prolapse. In the eye hospital in Colombo, where approximately 2,000 cataract extractions are performed yearly, it is claimed that the incidence of prolapse is 1.8 per cent less without iridectomy. In these hospitals iridectomy is done only when there is prolapse at the time of operation.

Expulsive hemorrhage occurs in about 0.5 per cent of these patients while on the table and is found in another 0.5 per cent at the time of first dressing. Of course, vision is always lost in these eyes.

Dislocation of the lens occurs so seldom with experienced operators, it is almost neglibible. However, many patients come in with dislocated lens which has floated up behind the pupil. Of course, these lenses have to be removed with the spoon. The lens couchers still do a big business in India.

Secondary cataract is a frequent complication following capsulotomy, and its treatment cannot be taken lightly. It necessitates puncture of the coats of the eye and is frequently followed by infection. In 86 needlings there was one infection. In 600 consecutive capsulotomies secondary cataract was found in 24.6 per cent at first dressing. Of course, some of these patients will have clear eyes in a few weeks, but since there is no way to follow these patients, the residual cannot be determined. In 400 consecutive intracapsulars, secondary cataract was found at first dressing in only 2 per cent. The tendency now is to remove the capsule with iris forceps instead of the conventional cutting or tearing operation.

Eversion of the cornea occurs in about 0.1 per cent.

Iritis is a frequent complication following capsulotomy but relatively rare following intracapsular. The percentage is approximately 15 and 2 per cent, respectively.

VISUAL RESULTS

As follow-up is impossible on the majority of these patients, it is impracticable to get any figures on the vision of the capsulotomy patients as compared with the intracapsular after a period of one, two, or more years. Very few patients are ever seen after they leave the hospital, and even while they are in the hospital the records are very meager and incomplete. If a patient can count fingers, his vision is recorded as good. No accurate refraction is done on the few patients who get glasses. A plus ten sphere is put on and if they say they can see better, that is what is prescribed. Perhaps 10 per cent of the operated patients get glasses.

CONCLUSION

1. More physicians in India use capsulotomy than the intracapsular method, but more cataracts are removed by the intracapsular than the capsulotomy method. There is no generally accepted technic in either method—each doctor is a law unto himself. (I saw about a dozen varieties of each method and even observed one man

who performed a canthotomy on each of his patients.)

2. Visual results, without second operation in a large percentage of these cases, are definitely superior in the intracapsular method.

3. With an experienced operator, loss of vitreous is about the same in either method but recovery is quicker and smoother in the intracapsular than in the capsulotomy method.

4. Expulsive hemorrhage occurs in about the same percentage of cases in either method.

5. Prolapse of the iris occurs more frequently in the intracapsular than the capsulotomy extraction.

6. Some surgeons claim that iridectomy increases the incidence of prolapse just as vehemently as the advocates of iridectomy claim that it reduces the incidence of prolapse.

7. With the skillful operator the intracapsular method seems to be the one of choice in uncomplicated, senile cataracts, producing results equal or superior to the capsulotomy method (except in the incidence of iris prolapse.)

8. One type of senile cataract is as easily removed by the intracapsular method as the other, provided the operator knows what type he is dealing with and makes his incision accordingly. There is greater vitreous loss when the conjunctival bridge is used due to the resistance offered to the passage of the lens.

9. Asepsis does not seem to play a very great part in the incidence of infection which is about the same in the better hospitals as in the camps.

10. One thing stands out clearly: The more experience a man has the less he complicates the operation by special instruments, unnecessary injections, questionable procedures and multiple operations such as preliminary iridectomies (other than in glaucoma), superior rectus suture, canthotomy, and so on.

THE PATHOLOGIC PHYSIOLOGY OF DIABETES MELLITUS*

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The average clinician can and does treat a diabetic patient adequately but, for the most part, on an empirical basis, having previously learned the routine to follow, the dosage of insulin to be administered, and the time of administration. However, the basic knowledge regarding this disease and its pathogenesis is severely lacking in these well-meaning physicians. It is true that even a Senior medical student might treat a diabetic in the routine manner and do a good job of it but, to support adequately and understand the condition in a patient, one must have the disease and its physiology within easy memory distance.

During the last month of my Junior year, while in medicine, I first realized my very inadequate knowledge of this condition and took upon myself the task of clearing up this situation in my own mind, and at the same time, write my Senior thesis. I apologize for the method of presentation, presenting no original work but actually a summary of a group of much more extensive articles by men who are responsible for present-day concepts of this interesting condition, diabetes mellitus.

I. *Normal Sugar Metabolism.* — Before we can discuss abnormal carbohydrate metabolism as seen in diabetes mellitus, a review of the normal must be considered.

A. *Digestion and Absorption.*—Following the ingestion of a carbohydrate meal, many reactions, not too well understood, take place. Wiggers[1] has prepared an excellent chart which shows graphically these changes.

All of the carbohydrate ingested soon assumes the form of monosaccharides. Of necessity, these must all later be converted to d-glucose by the liver in order that utili-

*Awarded the prize offered by the Professor of Medicine at Tulane University School of Medicine for the best thesis on a subject related to internal medicine.

zation of the carbohydrate may take place. It has been shown that no glucose is absorbed from the stomach.[2] The small intestine happens to be the site of the majority of the absorptive processes because of its greater length, large surface area, augmented by the 5 million tiny, active villi found as part of the wall, and the large lymphatic and blood vessel arrangement. Absorption is regulated, according to Best and Taylor,[3] by physico-chemical laws, such as osmotic pressure relationships between blood sugar and that in the enteric lumen, hydrostatic pressure of the lumen produced by intestinal contractions, osmotic pressure of the plasma proteins and cataphoresis (the flow of a substance from the anode to the cathode across a membrane). These laws cannot adequately explain carbohydrate absorption, in the light of experimental work done by Cori.[4] He showed the rate of absorption for each sugar to be constant, regardless of the concentration of that sugar in the enteric lumen. The rate for each sugar was shown to be different and, relatively, galactose, glucose, fructose, mannose, xylose, arabinose. Therefore, some vital activity is given to the epithelial cells of the intestinal villi. Absorption rates of the selective type have been shown to be stopped at a temperature of 0° C., at which temperature the sugars were absorbed purely in accordance with their diffusion rates, depending on the size of the molecule. According to Verzar and McDougal,[5] the absorption of glucose depends mainly on a phosphorylation process rather than an oxidative one.

B. *Distribution.*—Following absorption, the carbohydrate may go one of several pathways, (a) conversion to glycogen in the liver, striated muscle, cardiac muscle, brain, kidney, or other tissue; (b) oxidation by peripheral tissues: (c) conversion to tissue fats and stored as such; (d) retention in the blood stream for variable lengths of time, or (e) excretion with the urine by the renal mechanisms. About 75 per cent of the glucose absorbed from the intestine is converted to glycogen in the liver.[6] Glycogen is found in all cells but most abundantly in the liver, where it varies with the nutritive state of the individual.[7] Shipley and Humel[8] found this liver glycogen to be in two parts, one as glycogen alone and the other as a component of glycoprotein with blood glucose. The latter remains at a fairly constant level, any fluctuation occurring with the glycogen protein. Gylcogen found in the muscles, liver and other tissues of the body is known to be 10 times the amount of glucose found in the body of the human.[7] Since the blood glucose is the connecting link between the many processes, it seems only proper to consider this factor next.

C. *Blood glucose.*—Glucose is found to be evenly distributed between the cells and plasma of the circulating blood, indicating a cell permeability to this substance.[7] Somogyi[9] analyzed blood filtrates by several methods, following fermentation, and found 23-31 milligrams of non-fermenting reducing substance per 100 c. c. of blood. He found these substances, thought to be chiefly glutathione, ascorbic acid, and thioneine, to be for the most part, contained in the red blood corpuscles. The remaining 60-100 mg. per cent of reducing substance is fermentable and therefore glucose. Since the "saccharoid" level remains fairly constant, regardless of changes elsewhere, the total reducing value of the tungstic acid filtrate is clinically considered to be equivalent to the blood sugar.[10]

(1) *Origin of the blood glucose.*—As mentioned above, there is an exogenous source of sugar which is that absorbed carbohydrate from the gastrointestinal tract. There is also an endogenous source of blood glucose. Soskin[11] confirmed Mann's[12] original work, establishing the liver necessary for maintenance of the blood sugar. Hyperglycemic agents were shown to have no effect on the falling blood sugar seen in a liverless dog. Examination of the muscles of these dogs revealed a normal muscle glycogen, which Soskin considered enough evidence to conclude that no blood sugar was formed from muscle in the absence of the liver and that the liver was the source of glucose in the absence of eating or

other exogenous source. Cori,[13] in 1931, showed that muscle glycogen could contribute indirectly to the blood glucose by producing an excess of lactic acid, which diffused into the blood stream, was carried to the liver, and there converted to liver glycogen to be changed to blood glucose if necessary. Young[14] showed, following hepatectomy in dogs, a lowering of the blood glucose but no elevation of the lactic acid level. By actual measurements, he concluded that less than 10 per cent of the sugar was obtained from lactic acid. Therefore, muscle glycogen is used only for local needs and contractions. The absorption of glucose by the muscles is an irreversible process and the glucose absorbed is retained until burned regardless of the body's need. The glucose → lactic acid → liver glycogen apparently occurs only during strenuous work or anaerobic exercise. This will be discussed later in more detail.

(a) *Liver glycogen.*—Drury[15] has aptly said "The liver is to the blood sugar what the heart is to the circulation." Glycogen, according to Peters,[16] plays an administrative role, forming glucose when it is not needed elsewhere. Cori[18] in 1940, dialyzed liver extracts, removing the inorganic and organic phosphates and the coenzymes. When glycogen, inorganic phosphate, and adenylic acid were added, there was a breakdown of the glycogen seen with the formation of glucose. If either the inorganic phosphate or the adenylic acid was removed from the system, no glycogen breakdown occurred. With further experimentation, it was found that glycogen + H_3PO_4 ⇆ glucose — 1 — PO_4, a reversible reaction, requiring the enzyme phosphorylase for its initiation. The reaction is an anaerobic one, inhibited by phlorhizin, and requiring adenylic acid as coenzyme. This glucose — 1 — PO_4 is changed to glucose — 6 — PO_4 by the action of phosphoglucomutase. When either glucose — 1 — PO_4 or glucose — 6 — PO_4 is added to liver extract, glucose + inorganic phosphate is formed. The enzyme involved in the latter reaction is phosphatase. Therefore, the combined action of phosphatase, phosphoglucomutase, and phosphorylase is required for the simple reversible reaction: glycogen (liver) ⇆ glucose (blood). Kaplan and Greenberg,[18] using radioactive phosphorus, noted the changes in the acid soluble phosphates in the liver coincident with metabolic changes in carbohydrates. This work supported Cori's fairly well. Cori,[17] however, had previously noted an uptake of inorganic phosphates by the liver in the formation of glucose from glycogen. This reaction (glycogen ⇆ glucose) goes to the right in the presence of insulin deficiency, stimulation of the liver by epinephrine, thyroid hormone and others, and in hypoglycemia resulting from starvation. With complete insulin lack, as shown by Soskin and Levine,[20] the liver glycogen reserves are depleted. When this occurs, the body begins to compensate with the production of glucose from body protein via liver glycogen.

(b) *Protein.*—If the carbohydrate stores of the body become depleted, protein takes over the job of supplying the carbohydrate needed. If only decreased carbohydrate is present, the body uses very small amounts of it and only when necessary.

In 1890, Mering and Minkowski, using a depancreatized dog, showed glycosuria and quantitative excretion of any administered sugar. They concluded that no carbohydrate was being utilized. They found 2.8 grams of dextrose excreted in the urine for every 1 gram of nitrogen and established the present D/N ratio. Soskin[21] criticizes their technic and choice of animals for determining this ratio. He points out that a wide variety of D/N ratios were seen among the different animals used and also in the same animal, depending on the number of days since pancreatectomy and condition of the animal. Other experimentation, headed by Lusk[22] repeated the experiments and found the D/N ratio to be 3.6 instead of Minkowski's 2.81. Lusk went further by saying if there were 3.6 grams of glucose in the urine to every 1 gram of nitrogen, then $\frac{3.6}{6.25} \times 100 = 58$ grams of glucose for every 100 grams of protein ingested. Soskin[21] con-

cluded that sugar was derived partly from protein but considered it impossible to say to what extent it occurred since some fat might have been converted to sugar also. Shaffer[23] considered the glucose ingested as 100 per cent CHO + 58 per cent P + 10 per cent F. Ten per cent of the fat weight is in the form of glycerol and this portion is or can be converted to glucose.[24] Young[14] gave fat to depancreatized and fasting animals and noticed no elevation of the blood sugar or glycosuria or rise in liver glycogen. While the possibility exists that the animals used were already using their maximum amounts of fat in producing blood glucose, Drury says that while it may be possible, the experimental evidence has yet to be shown. Stadie[25] concurs with Drury in saying the diabetic picture can be explained without the necessity of an hypothesis similar to this: that fat → carbohydrate.

Having reviewed some of the experimental evidence, we can say that, in the normal individual, the blood glucose is supplied entirely by the liver and any exogenous means which may be used. However, when this liver is depleted of some or all of its glycogen, the body supplies of protein "pitch in", so to speak, in an attempt to keep the body well supplied with glucose. The possibility of any of the fat molecule, excepting the glycerol fraction being converted to glucose is not yet shown to be an actual process.

(2) *Utilization of the blood glucose.*—The glucose present in the blood stream may be utilized, that is, disappear from the blood stream and extrahepatic tissues, by one of the following four means:[7]

(a) *Formation of glycogen.*—This may take place in the liver or muscles of the body. It has been demonstrated that the glycogen content of the liver increases following a carbohydrate meal.[14] Kostelitz and Ritchie[26] found the glycogen content of the liver to increase with glucose administration and insulin injections. The phosphorus content of the liver was similarly increased when the glycogen and glucose levels were raised. Mann[13] found that only glucose could be utilized by the peripheral tissues, all other carbohydrate having to be changed to glucose or a polymer by the liver. Therefore, since glucose will be used by both liver and tissues, while the others are used only by the liver, the glucose should disappear faster, as it has been shown to do. The process of glucose ⇆ glycogen has already been mentioned but I will again show it to impress further these reactions upon the reader. Glucose + inorganic phosphates, catalyzed by phosphatase → glucose — 6 — PO_4 → glucose + PO_4, with the aid of phosphoglucomutase → glycogen, catalyzed by phosphorylase. This reaction occurs in both liver and muscle, where glycogen is mostly laid down. Dorfman[26a] states that while evidence does not rule out other pathways, they are unnecessary to explain the facts. Formation of muscle glycogen differs from that in the liver by being an irreversible process in the muscle, while in the liver, the glycogen ⇆ glucose relationship is easily reversible by a number of factors. The glycogen in the muscle is removed only by oxidation, aerobic or anaerobic.

(b) *Oxidation in the tissues.*—This process may occur either with or without oxygenation. In 1907, Fletcher and Hopkins[27] working with frog muscle under anaerobic conditions, noted an increase in lactic acid in the muscle. Embden[28] later said that the lactic acid played no part in contraction of muscle and that it was formed after contraction was over as well as during contraction. Meyerhof[29] argued this point with him, saying it was essential to the muscle contraction and was the only source of energy during work. Eggleton and Eggleton discovered creatine phosphoric acid, Meyerhof and Lohmann[30] later showing that the breakdown of this substance in active muscle gave off heat. Lundsgaard,[31] in the early thirties, used iodo-acetic acid, which inactivated the enzymes necessary for lactic acid formation. The muscles examined under that condition were able to do work but less than the normal muscle; so the lactic acid was shown not to be essential to contraction but necessary for an energy reserve. Along with these same conditions, it was noted that the intensity of the break-

down of creatine phosphoric acid was directly related to the amount of work done until exhaustion took place, at which time, another substance, adenylic phosphoric acid, began to break down. Further experiments were done, most of them by Lundsgaard and his co-workers, until some definite conclusions were reached. The breakdown of creatine phosphoric acid, releasing about 0.3 calorie per gram of muscle, was greatest in the earliest part of contraction, gradually decreasing through a series of contractions. On the other hand, lactic acid formation from glycogen, liberating about 1.0 calorie per gram of muscle, was very minute at first and increased in breakdown as the contractions were continued. This showed Embden to have been right. The energy produced by each substance tended to balance with a common amount of energy throughout the muscle contraction. Lundsgaard[31] relates the reaction in the following way: Adenylic phosphoric acid breaks down (cannot be demonstrated in humans) and in some way is related to the initial constriction of muscle. Following this, creatine phosphoric acid breaks down, the energy liberated producing a resynthesis of the adenylic phosphoric acid from its breakdown products. As the breakdown of creatine phorphoric acid decreases, lactic acid formation is taking place and producing enough energy to resynthesize creatine phosphoric acid from the creatine and phosphoric acid present. The lactic acid formation continues until muscular action ceases. One must remember that the above reactions, with lactic acid formation, take place in anaerobic conditions. As soon as oxygen is added, the situation changes. Fletcher and Hopkins[27] showed this when oxygen was added to the anaerobic frog muscle. The lactic acid which had accumulated disappeared. Lundsgaard[32] said, "No reason can be found for accepting Meyerhof's interpretation of the Pasteur-Meyerhof reaction in aerobically working muscles .... still speak of the Pasteur Meyerhof reaction in aerobically working muscle, for, by this, we merely understand that the formation of lactic acid recedes in the muscles under aerobic conditions." It has been generally assumed that aerobically working muscle metabolizes carbohydrate only, but we know that heavy exercise may be performed on a fatty diet alone and with a "fatty" respiratory quotient.[32] Possibly fat is being used directly or is converted to carbohydrate and used.

According to Meyerhof,[33] under aerobic conditions, no lactic acid is formed, the pyruvic acid being completely oxidized to $CO_2 + H_2O$. One more glance at the chart will reveal that pyruvic acid and not lactic acid is the intermediary substance of most importance. The lactic acid formed, just to clear up this point, is resynthesized to glycogen but not in the muscle where it is formed. It must be carried by the blood to the liver where it is changed to glycogen.[13]

(c) *Storage as fat.*—Peters[16] shows where carbohydrate can form any of the fatty acids with one exception, that being small quantities of unsaturated long-chain fatty acids. Once converted, it may be stored in anhydrous form and having potentially 9 calories per gram. When needed for energy, they need not be reconverted to glucose but may proceed to the liver, change to ketone bodies, and be oxidized in a metabolic cycle in the peripheral tissues, mainly the muscles.

(d) *Excretion by the kidneys.*—Cori[34] found glycosuria to be present in animals only after enough sugar had been absorbed to raise the concentration of the sugar in both the blood and tissues to a certain level. The renal threshold for glucose varies somewhat with the individual but the average quoted figure is 160 ± mg. of glucose per 100 c.c. of blood. Any level over the amount results in glycosuria. John[35] mentions that the threshold may vary any place from 65 to 450 mg. per cent. He brought out a rather interesting point, that it is difficult for the glucose to pass the renal threshold but, once the penetration has begun, it continues even with a level of blood sugar below the normal threshold.

(e) *Respiratory quotient.*—Since many investigators have used the R.Q. as evidence of carbohydrate and fat metabolism, we

should consider it and evaluate it. The R.Q. is the ratio of $\frac{CO_2 \text{ expired}}{O_2 \text{ inspired}}$ and, according to theory, equals one if pure carbohydrate is being utilized. If one molecule of carbohydrate is oxidized ($C_6H_{12}O_6 + 6\ O_2 \rightarrow 6\ CO_2 + 6$ HOH) one can see that the amount of oxygen breathed or utilized is equal to the amount of carbon dioxide excreted. In the completely depancreatized animals, the R. Q. is in the neighborhood of 0.7, which many take to indicate pure fat metabolism, since more O_2 is required for the oxidation of one molecule and, therefore, the R. Q. is lowered. Young[14] points out that sugar is being utilized to some degree, even in the depancreatized animals, and, that being the case, the R. Q. is only a resultant of two oxidative processes. And, if the R. Q. of the depancreatized animals is 0.7, with small amounts of glucose being oxidized, the actual R. Q. of fat must be less than 0.7. Young therefore suggests we reconsider our interpretation of the R. Q. Soskin[21] goes further than this, stating that the R. Q. is only an index for the whole body. The brain can utilize only glucose, so its R. Q. must be 1.0. Therefore, any other substance in the body, giving an R. Q. lower than this will not be shown to any accuracy by the overall R. Q. which is what is determined. Soskin mentions that oxidative processes alone do not make up the R. Q. but that many transformations and resyntheses and partial oxidations, all taking place in the body, have definite effects on the total R. Q. So, the true R. Q. is only an equilibrium among all the metabolic processes in the body. Werthessen[36] showed this by training rats to eat their 24 hour food requirement in one to five hours and found their R. Q. to vary from 0.27 to 1.70. He apparently caught the metabolic processes "with their pants down", so to speak, before the equilibrium of 0.7 — 1.0 usually found could be established.

(3) *Control of the blood sugar levels.*—Relative changes in the production and utilization of the blood glucose may occur and one can easily see the effect on the level of glucose in the blood. Definite factors produce these changes.

(a) *The blood sugar level.*—There is definite evidence to show that the level of sugar in the blood stream is one of the most important determining factors in the control of its own level. Soskin and Levine[20] have shown that carbohydrate utilization in the peripheral tissues, in the absence of insulin, increased proportionately to the elevation of glucose in the blood stream. In a normal individual, with normal amounts of insulin, an increase in the blood sugar level will decrease the output of glucose from the liver and vice versa. In addition, an increase in blood sugar causes the reaction glycogen $\leftrightarrows$ glucose to move to the left and vice versa. Upon these facts is the glucose tolerance test based. It was shown by Soskin, Allweiss and Cohn[37] that the decrease in the glucose blood level following the initial rise was not due to an increased output of insulin with increased utilization. They depancreatized animals, administered constant amounts of glucose with the necessary amount of insulin required to keep the blood level constant and then performed a glucose tolerance test. The test revealed a normal curve, indicating insulin stimulation was not a factor in this test. However, if the liver was removed instead of the pancreas and the experiment repeated, a diabetic curve was obtained. This showed conclusively that the liver's conversion of glucose $\rightarrow$ glycogen, the inhibition of the reaction glycogen $\rightarrow$ glucose, and increased utilization of sugar by the peripheral tissues were all on the basis of the increased blood sugar. Insulin in certain amounts was found necessary for the reactions to occur properly,[38] but no increase over this certain amount was needed, the stimulus being the high blood sugar level alone. Further work, quoted by Soskin,[21] supports the assumptions mentioned below:

(a) Small amounts of added glucose $\rightarrow$ inhibit glucose output by the liver.

(b) Moderate amounts $\rightarrow$ inhibit glucose output and increase glycogen formation in the liver and muscles.

(c) Large amounts → inhibit glucose output, increase glycogen formation, increase utilization of glucose by the tissues, and increase peripheral storage of glucose in the form of fat.

We can summarize the above data by saying a definite relationship exists between the blood sugar level and production on one hand and between the level and utilization on the other.

(b) *Insulin.*—In the absence of insulin, Soskin[21] states that no decrease in the rate of sugar utilization by the extrahepatic tissue occurs, if the blood sugar level is high enough. The liver, in an attempt to keep this blood level high, shows an increase in glycolysis with an increased glucose production and decreased glycogen content. With the decrease in liver glycogen, an increase in gluconeogenesis occurs with further changes. Administration of insulin will stop all of these processes and revert the metabolism back to normal. How does it work? Insulin administration will lower the blood sugar, increase the muscle glycogen and, depending on the blood glucose level, will raise or lower the liver glycogen. Cori and Cori[39] showed that the increase in glycogen in the muscle did not occur in the adrenalectomized animal and attributed the increase to a stimulation of adrenalin by the insulin, with adrenalin's effect causing the increase in muscle glycogen. Soskin and Levine[20] show that insulin is not needed for carbohydrate utilization, which can continue normally if the blood sugar is high enough, so insulin cannot produce an increased oxidation. But they say insulin is a catalyst, allowing the same amount of oxidation to proceed at a much lower blood glucose level. This sounds fairly reasonable. Lavietes[40] shows that, where the blood level was previously high, insulin will decrease glucose formation by the liver, thereby enhancing glycogen deposition in the liver. In the case of insulin administration to the normal person, a low blood sugar will result with decreased peripheral oxidation and glycolysis will be stimulated. Therefore, the process glycogen ⇆ glucose, according to Lavietes is secondary, not to the blood glucose level primarily but to oxidation of carbohydrate in the peripheral tissues.

Soskin[21] concludes that, since glucose utilization and glycogen deposition can occur in the depancreatized as well as the normal animal but at a slower rate for that blood level and at a normal rate if the blood level is elevated, insulin simply modifies the relationship between sugar concentration and sugar utilization rates. Lavietes[40] points out that insulin does not inhibit the liver glycogen → glucose reaction directly but by increasing the oxidation of carbohydrate at a lower level of blood glucose, the need for the high level, produced by glycogenolysis in the liver is lessened, allowing gluconeogenesis to surpass glycogenolysis with a resultant increase in liver glycogen. The increase in muscle glycogen seen following insulin administration is thought to follow the increase in liver glycogen, producing a lessened need for gluconeogenesis from protein and thereby stopping the breakdown of muscle glycogen. It must be kept in mind that a therapeutic paradox may occur if too much insulin is administered,[40a] decreasing the blood glucose level, which stimulates the liver to glucose production and thereby starting the processes forward again, with a decrease in liver glycogen, increased gluconeogenesis, and decreased muscle glycogen. Shipley and Humel[8] noted in liver slices, with insulin administration, a decrease in the liver glycogen and an increase in glucose output. They concluded that insulin accelerates the glycogen → glucose reaction. But, this being *in vitro*, it seems more likely that the reaction may proceed in either direction depending on the amounts of carbohydrate oxidized in the peripheral tissues, which in turn depends on the blood level and the presence of insulin. Stadie[41] thinks insulin aids in the phosphorylation of glucose which makes it easier for the tissues to store or utilize it. Soskin[20] definitely states that insulin does not promote the oxidative breakdown of glucose but is primarily concerned with the regulation of the blood sugar level, affecting the output of sugar from the liver and its rate

of uptake but not oxidation by the tissues. Summing up this evidence, one can say that, according to the present views, which are still incomplete, insulin is a cataclyst, which is necessary for the rapid phosphorylation of glucose to glucose — 1 — PO_4, the presence of which easily facilitates tissue oxidation and glycogen production in muscle and liver. These processes of tissue utilization and glycogen production may continue in the absence of insulin but require much higher blood sugar levels than when insulin is present. And, lastly, all of the other effects of insulin can be explained adequately on the basis of this increased phosphorylation process alone.

Haist[42] has shown that the insulin content of the pancreas is decreased in two different and opposite conditions: (a) fasting, fat feeding, and insulin administration will, by relieving the need for insulin, make the cells less active. Insulin administration in this condition will further reduce the content. (b) Partial pancreatectomy or injection of anterior pituitary extracts into dogs will, by increasing the blood sugar level and keeping the level high, stimulate the beta cells of the pancreas to exhaustion with later atrophy and degeneration. Insulin administration in this type would prevent the pathologic changes occurring.

Stimulation of the beta cells is said to result[42] from high blood sugar levels, low blood insulin levels, a minor effect produced by the central nervous system, and from stimuli originating in the anterior pituitary gland.

Factors other than those mentioned above are of importance in regulating the normal blood sugar level. Long[43] brings out the fact that there are hormonal influences in the body which influence processes concerned with glucose utilization and those which affect the formation of glucose and other carbohydrates from non-carbohydrate sources. These factors will be considered below.

(c) *Influence of the anterior pituitary gland.*—Many experiments, in regard to the effect of the anterior pituitary upon sugar metabolism, have been carried out. In a depancreatized animal with typical diabetes mellitus, the removal of the anterior lobe of the hypophysis will produce an almost complete remission of the signs and symptoms previously noted.[43-46] The hyperglycemia, ketosis, acidosis, and glycosuria disappear, while the nitrogen excretion by the kidneys is reduced, the elevated D/N ratio is lowered, and the survival time of the animal is lengthened. The anterior pituitary, apparently, in the absence of the pancreas, promotes either an increased production of sugar or a decreased utilization or both.[46] Therefore, the removal of the gland was very similar in many ways to the administration of a dose of insulin. The above shows that, with the removal of the organ there occurs a decrease in tissue breakdown to glucose or an increased utilization of the carbohydrate.

If the anterior pituitary is removed in a normal animal[43] the sensitivity to insulin administration is greatly increased, there occurs an increased resistance to the hyperglycemic action of epinephrine, glucose absorption is retarded, and glucose tolerance is retarded or normal, the latter depending on the animal used. In addition to these effects noted by Long[43], Houssay[44-46] mentions a decreased nitrogen excretion by the kidneys, hypoglycemia, a marked decrease in liver glycogen, and no change in muscle glycogen in dogs and rabbits. Here again the effects seen may be explained either on the basis of decreased breakdown of tissue or increased carbohydrate utilization.

If the anterior lobe extract is injected into normal animals,[46] a hyperglycemia and glycosuria occur if the animal has been previously well fed; nitrogen excretion in the urine increased, the R. Q. decreases, and the animal's sensitivity to insulin is greatly reduced. A postmortem examination of the animal will reveal a certain amount of degeneration in the islets of Langerhans of the pancreas. To explain these actions of the anterior pituitary extracts, we must consider other information from further experiments: (a) If the liver is removed before the anterior pituitary extract is injected, the hyperglycemia is not seen.[43]

A previous section of the splanchnic nerves will produce the same effects as hepatectomy. The liver, therefore, is stimulated in some way, possibly through the sympathetics to produce more glucose from glycogen and is directly responsible for the hyperglycemia seen following an injection of anterior pituitary extract. (b) The R. Q. is decreased following an injection of anterior pituitary extract. Long takes this to mean that carbohydrate utilization by the tissues is decreased. (c) The increased nitrogen excretion seen above is thought to be indicative of an increase in protein breakdown by the body. So, apparently, the anterior pituitary extract stimulates glucose production in the liver, decreases carbohydrate utilization by the tissues, and increases protein breakdown in the body. Houssay[46] gives the anterior hypophysis four actions in regard to carbohydrate metabolism: (a) hypoglycemic, (b) hyperglycemic, (c) glycogenetic, and (d) glycogenolytic.

It is possible and probable that this gland acts directly on carbohydrate metabolic processes or is it more probable that this action is extended to other organs in the body? The part the liver plays has already been mentioned. Long,[43] using dogs, showed the glycosuric and ketonuric effects of the injection of anterior pituitary extract. These were not prevented by the previous removal of either the thyroid or adrenal medulla but were definitely inhibited when the adrenal cortex was removed. If, therefore, this action is carried out by the stimulation of the adrenal cortex, injection of adrenocortical extracts should produce similar effects in the metabolism of glucose.

(d) *Adrenal cortical influence.*—If adrenocortical hormone (Kendall's Compound, E, C-11-17 oxygenated adrenal cortical steroid) is injected into a normal animal, the blood sugar increases, glycosuria results, and large amounts of nitrogen are extracted by the kidneys.[43, 47] If an experimental animal is made diabetic by pancreatectomy and then, following this, a bilateral adrenalectomy is performed, the effect is almost the same as if the anterior pituitary gland had been removed, that is, a remission of symptoms and general metabolic disturbances occur.[43] This will not happen if only the medullary portions of the glands are removed. These effects are thought to be due to an increase in protein catabolism with gluconeogenesis since (a) urinary nitrogen rises before urinary sugar excretion does and (b) the blood sugar level is much higher than would be expected if only due to an inhibition of carbohydrate oxidation or increased production of glucose from the liver.

The weight loss and urinary excretion products have been compared between a case of pancreatic and one of adrenal diabetes.[47] The changes were found to be about the same but, whereas insulin injections cured the pancreatic case, it had no effect on the adrenal case except in extremely high doses. Therefore, this "sugar hormone" of the adrenal cortex is theoretically directly antagonistic to insulin, thereby decreasing carbohydrate utilization and stimulating to the breakdown of protein, with an over-production of nitrogen and glucose resulting. The protein mainly affected is thought to be that contained in the lymphoid tissues of the body.

The pituitary gland, by acting through the adrenal cortex, produces the same effects plus the effects resulting from its stimulating action on the liver, increasing glucose production. The degeneration of the pancreatic islets seen following an injection of the anterior lobe extracts is thought to be due to an exhaustion atrophy, secondary to the sustained hyperglycemia present.[48]This pathologic process is preventable by a previous or simultaneous injection of insulin with the pituitary extract, thus indicating no direct toxic effects on the beta cells by the hypophyseal hormone.

(e) *Others Factors Involved*

(1) *Thyroid hormone.*—The effect of the thyroid as seen in cases of hyperthyroidism is to produce a hyperglycemia and glycosuria. Joslin and Lahey[49] found a diabetic glucose tolerance curve and glycosuria in about one-third of a series of hyperthyroid patients in 1943. This effect was found to

be absent in experimental animals following hepatectomy or a splanchnic section indicating an action on the liver, increasing glucose production. Althausen et al.[50] noted an abnormal galactose tolerance curve in hyperthyroid patients following oral administration of the sugar but a normal response to an intravenous injection. This led him to say the action of the thyroid hormone was on the phosphorylation mechanism in the intestional mucosa, increasing its action and thereby glucose absorption in cases of hyperthyroidism. No direct effect on the metabolism, other than a generalized increase in rate, could be found.

(2) *Adrenal medullary hormone.*—Injections of this substance produce a hyperglycemia secondary to an increased mobilization from the liver.[3] Muscle glycogen has been shown to decrease, raising the blood lactic acid level.[14] The lactic acid is carried to the liver, where it is converted to glycogen, later being changed to glucose (Cori cycle). Small amounts of adrenalin will decrease liver glycogen, slightly larger doses will decrease muscle glycogen and increase liver glycogen, while large doses reduce both the liver and muscle glycogen contents. This hormone is not thought to act except in exercise and emotional disturbances.

(3) *Posterior pituitary gland.*—This organ was thought by Burns in 1923 to have an antagonistic action to insulin since no change in the blood sugar level was noted when posterior pituitary extract was injected subcutaneously with insulin. Griffith[51] repeated these experiments and used the intravenous route in addition, noting a normal insulin response. He concluded the effect of the posterior pituitary extract to be local, causing marked vasoconstriction and thereby preventing absorption of the insulin.

(4) *Diet and nutrition.*—Blatner and Murphy[52] noted a lowering of the blood sugar following the injection of 180 grams of whole fresh liver equivalent to the effect seen with 10 units of insulin. Biskind and Schreier[53] noted the same effects but related them to the vitamin B complex contained in the liver used. They mentioned other works, showing fairly well that vitamin B1, along with riboflavin niacin amide, ascorbic acid, vitamin A and vitamin D, are all essential to carbohydrate metabolism. When the liver is damaged by a toxic drug, a diabetic glucose tolerance curve can be obtained.[54] Since the conversion of blood glucose → liver glycogen and the reverse depends on enzyme systems in the liver, any dysfunction of these enzymes will bring about changes in carbohydrate metabolism, expressed as hyperglycemia, glycosuria, and a delayed glucose tolerance curve.[55] This type of dysfunction is seen in cases of obesity with fatty livers and in patients with vitamin B deficiency.

(f) *Experimental work with carbohydrate metabolism.*—In addition to the large amount of experimental work mentioned previously, there has been some good work done recently which should be at least mentioned in a paper on carbohydrate metabolism and diabetes.

(1) *Phlorhizin.*—Following the administration of this substance, a glycosuria, hypoglycemia and decrease in liver glycogen all occur, in addition to other effects. This specific action is thought to be on the phosphorylating mechanism so essential to carbohydrate metabolism. By inhibiting this mechanism, phlorhizin prevents intestinal absorption of glucose, slows down or stops the glycogen glucose processes throughout the body and inhibits glucose resorption by the renal tubules. Lukens, Dolens, and Wolcott, quoted by Lukens[56] compared the effects of insulin with those of phlorhizin as is shown by the table on page 461.

(2) *Alloxan.—This* substance, which is an oxidation product of uric acid and, as yet, not known to be present in the human intermediary metabolism of uric acid, has been found to be definitely diabetogenic in character. A considerable amount of experimental work has been done with this substance in an attempt to learn something further about diabetes mellitus, and related problems.[48, 57, 58, 59, 60, 61] Goldner[48] presents an excellent summary of the work on this substance up to January, 1945.

Phlorhizin	*Insulin*
A. *Similar Effects*	
1. Reduction of hyperglycemia	
2. Restoration of hydropic islands of Langerhans	
B. *Different or Opposite Effects*	
1. Impairs resorption of glucose by kidney	1. No such action
2. Increases protein catabolism (N excretion)	2. Decreases protein catabolism (N excretion)
3. Increases ketogenesis	3. Decreases ketogenesis
4. Amount of food assimilated by diabetics is increased, decreased, or unchanged.	4. Food assimilation in diabetics is increased
5. Slight gain or loss of weight occurs	5. Marked gain in weight occurs
C. *Uncertain Effects, Common Assumptions listed*	
The following are probably secondary to a loss of glucose:	The following may include the primary action of insulin:
1. Decreased carbohydrate oxidation	1. Increased carbohydrate oxidation
2. Liver and muscle glycogen is usually decreased but may be increased by adding glucose or insulin	2. Increased liver and muscle glycogen in the diabetic.

Following an injection of alloxan in diabetogenic doses, a typical diabetes mellitus becomes evident in twenty-four to forty-eight hours, including polyphagia, polydipsia, polyuria, hyperglycemia, glycosuria, and weight loss. During the first twenty-four hours, however, there occurs a marked hypoglycemia with death, if glucose is not administered. If insulin administration is continued, animals may live and remain well for many months. Without insulin, they lose weight, become weak, very susceptible to infection, show hyperglycemia, hyperlipemia, fatty degeneration of the liver and death in six to seven weeks. Depending upon the animal used, ketosis, acidosis, cataracts, and retinitis may be demonstrated. Postmortem changes noted are beta cell necrosis in the islets of Langerhans with no evidence of local inflammation and, with large doses, fatty infiltration of the liver and glycogen deposition in the renal tubules. Degenerative changes in the basophilic cells of the anterior lobe of the hypophysis with some necrosis of the cells in the adrenal cortex have been noted by Thomas and Emerson.[57] The latter pathological changes were not prevented by previous adrenalectomy nor could similar changes be produced by injecting large amounts of adrenocortical extract into the animals. So, these changes were not secondary to hyperglycemia but appeared to be a direct result of the alloxan.

The insulin content of the pancreas per gram of tissue is decreased following alloxan administration.[48] The hyperglycemias seen following injections of anterior pituitary extract occurred before islet cell degeneration took place and the degeneration was therefore thought to be secondary to the increased blood sugar level, being prevented by concurrent insulin administration.[48] This is not the case with alloxan diabetes, in which the degenerative changes are not inhibited or prevented by concurrent insulin administration. Therefore, a direct toxic action of alloxan on the pancreatic islets must exist. If the animal has been previously adrenalectomized, the symptoms

of alloxan diabetes disappear or fail to appear.[58] Apparently gluconeogenesis must be stimulated by the administration of alloxan.

The hypoglycemia seen in the early part of the syndrome is not seen if the animal has been previously depancreatized.[48, 59] Therefore, either the islets are stimulated to exhaustion and later atrophy or the degenerative processes release a large number of insulin previously formed within the cell walls. Since the beta cell degeneration occurs before the hypoglycemia, the second theory is supported by Goldner. Bannerjie[59] reviewed the literature on the subject, and with his experimental results, concurred with Goldner in thinking the hypoglycemia to be due to the process of degeneration with the release of preformed insulin followed by hypoglycemia. Once this phase has passed, however, the animal remains in a constant state of hyperglycemia as a result of the insulin lack now present.

(3) Thorogood and Zimmermann[60] bring out the idea of a second pancreatic hormone which opposes insulin, raising the blood level of sugar, promoting glycosuria, and preventing ketosis and coma in insulin deficient animals. Some interesting work has been done on this possibility, which we will not have time to discuss.

II. *Metabolic Disturbances as Seen in Diabetes Mellitus*

Diabetes mellitus is defined as a disturbance in carbohydrate metabolism secondary to a deficiency in the insulin supply of the body.[43] This may be an actual or relative deficiency but this paper is concerned only with an actual one. First, a word about the pathology associated with this disease. Long[43] noted a disproportion between the severity of the disease process and the degree of destruction seen in the pancreas at postmortem examination. Gomori[62] found no changes in some seven cases of long duration and marked changes in occasioned non-diabetics. To produce glycosuria in a dog, a least nine-tenths of his pancreas must be removed. Warren,[63] in 1937, mentioned that regardless of the severity of the disease, no prediction could be made with any degree of certainty as to what would be found at autopsy. He said that at least one-fourth of the pancreases showed no appreciable pathology. Engelhardt and Bruno,[64] in a series of 101 cases examined at autopsy, found only 17 pancreases with any pathologic findings. Warren and Root[65] summarized the pathological findings thusly, "Whatever the cause may be, it seemingly acts over a long period of time, perhaps throughout the duration of the disease. The pathology which we find in the pancreas at autopsy rarely represents the initial damage to the organ, but rather the result of a long struggle between the regenerative activity of the pancreas and the degenerative changes caused by the diabetogenic factor. The pancreas is not a static organ like the brain or myocardium, unable to repair itself after injury. . . . believe that this unfavorable course of the disease is not due to failure of the pancreas to regenerate but to continued injurious action on the organ by the causal agent, eventually overcoming the regenerative efforts."

If one understands in detail the normal metabolic processes associated with carbohydrate metabolism and the effects of administering insulin in animals and humans, the changes seen with insulin deficiency should be indeed obvious.

(A) *Overproduction vs. Underutilization.*—Since the level of the glucose in the blood stream depends on two factors mainly, that is, production and utilization, a hyperglycemia could result from an increased activity of the first facor or a decreased acivity of the second. The two theories regarding diabetes mellitus are classified in the same way, one saying the disease is a result of the inability of the body to utilize glucose in the absence of insulin and the other claiming that glucose is being utilized in the normal manner but that too much carbohydrate is being produced in the absence of insulin.

Minkowski first began the underutilization argument. Lusk and his co-workers said the completely depancreatized dog could not utilize glucose. Their beliefs were based on the following experimental re-

sults: (a) The non-protein R. Q. of diabetics was at a level precluding carbohydrate oxidation.. (b) Any administered sugar was quantitatively excreted by the kidneys. (c) An increase in the arteriovenous difference of blood sugar levels in muscles in a normal animal after glucose administration was diminished or absent in a diabetic. With these facts proved, the overproduction school had to say that carbohydrate was produced from fat in the body and that being the case, the R. Q. would only be an average between the R. Q. of fat → sugar in the liver (less than .7) and the R. Q. of sugar being metabolized in the peripheral tissues (more than .7). Soskin's[21] remarks about the R. Q. make it rather poor evidence for or against any theory. And, since the body was working with the maximum amount of glucose it would handle anyway, no wonder that the A-V difference did not increase or quantitative excretion of ingested carbohydrate take place.

Mann and Magath[66] in 1913 demonstrated that the removal of the liver from the depancreatized dog resulted in the development of hypoglycemia at the same rate and to the same degree as in the normal dog. Mirsky[67] found this to be true in his experimental work. Yater, Markowitz, and Cahoon[68] found carbohydrate oxidation to be occurring in a diabetic but at a subnormal rate, however. Soskin and Levine[20] showed that a diabetic animal with a hyperglycemia could metabolize the normal amount of glucose a non-diabetic could at a normal blood sugar level. Therefore the overproduction theory seems more tenable at the present time.

The source of the extra glucose has been shown to be the liver and through it, various pathways of gluconeogenesis. It is possible that glucose may be formed from protein and fat deposits in the body. Most authorities in general agree that protein may be converted to glucose but it is still a controversial point; the results indicate the formation of glucose from fatty acids and ketone bodies. Stadie[25] believes in the overproduction of sugar as a result of the body's inability to utilize sugar to its fullest capacity but definitely is against any such mechanism producing glucose from fat. He claims that the extra energy needed is obtained from the utilization of fatty acids by the body directly, without conversion to glucose being necessary. Yater, Markowitz, and Cahoon[68] combined both theories, which seems reasonable, and said the diabetic animal cannot utilize glucose as well as the normal one and in some way stimulates the liver to overproduce glucose from glycogen. When the liver glycogen stores become depleted, gluconeogenesis from lactic acid and protein takes place. The conversion of fat to glucose is still unproved.

B. *Hyperglycemia.*—Accepting the last few statements and again referring to Soskin and Levine,[20] one can see what might be expected to occur in the presence of a lack of insulin. The tissues of the body cannot utilize glucose at the normal blood sugar level and, in some way, stimulate the liver to produce larger amounts of glucose, even with a normal or elevated blood glucose present. This elevation in blood glucose, if high enough, enables the tissues to utilize the carbohydrate normally. With the decreased amount of insulin present, the inhibiting action of insulin on glucose production by the liver is also reduced, and glucose is produced freely. The liver continues to supply glucose to the blood stream with little or no replacement of its depleted glycogen. When a certain point is reached, however, gluconeogenetic processes are speeded up in an attempt to replace the liver glycogen from protein and lactic acid stores in the body. As a result of the increased protein breakdown to form glycogen in the liver and the muscle glycogen → lactic acid → glycogen in liver, there occurs a marked weight loss. Mirski[67] has shown that insulin decreases the rate of glyconeogenesis from protein. Polyphagia is probably a result of the increased weight loss seen. There is also occurring a depletion of the fat stores of the body and will be considered later. The R. Q. decreases because less glucose is being oxidized at the exsiting

blood sugar level and more energy is being used for transformative and partially oxidative reactions.[21] The D/N ratio is elevated since more sugar is being eliminated by the kidneys than normally and catabolism of protein has been speeded up.

C. *Glucose Tolerance Test.* — The one hour two dose test of Exton and Rose[69] seems to be the most sensitive and accurate diagnostic test for diabetes mellitus. A dose of dextrose is given and repeated in thirty minutes, while urine and blood samples are being taken. A normal test shows a normal fasting blood sugar level (80-120 mg. per cent), a rise not to exceed 75 mg. per cent in thirty minutes, the same, less, or not greater than 5 mg. per cent rise in sixty minutes over the thirty minute sample, and all urine samples negative to Benedict's test. These results depend on the body's ability normally to convert glucose to glycogen in the liver and the muscles as shown by Soskin et al.[37] Normally, with more glucose added, more glycogen will be formed. Insulin was shown to be necessary in certain amounts for this reaction but did not increase significantly following glucose administration to bring about increased glucose oxidation, as many had previously believed. In a diabetic, with a decreased supply of insulin present, the conversion of glucose → glycogen is not as rapid as normal and not as complete at certain blood sugar levels. Therefore, with the Exton Rose test, we see a normal or high fasting level, a rise of greater than 75 mg. per cent in thirty minutes, a rise of not less than 10 mg. per cent in the sixty minute sample over the thirty minute one, and glycosuria in one or more of the urine samples.

D. *Glycosuria.*—Glucose is a "high threshold" substance for the renal mechanism, is normally absorbed with water to a large extent, and consequently is absent from the urine. This reabsorption process is an active one, occurring in the proximal renal tubules as a result of phosphorylation of the glucose molecule by kidney phosphorylase and dephosphorylation by alkaline phosphatase in the tubular cells.

When the blood sugar concentration increases above certain normal limits (about 160 mg. per cent) the active resorption process is slowed or stopped. In addition, the increased concentration of glucose in the glomerular filtrate causes the osmotic pressure at which the reabsorption process ceases to be reached sooner than in the normal kidney.[3] In the loop of Henle this concentration of glucose exerts an osmotic pressure and thereby brings water back into the tubules, increasing the amount of water lost by the body and producing a diuresis.[70] Johns[35], by means of comparing simultaneously drawn blood and urine samples, noted that glycosuria occurred at higher levels on the ascent of the blood tolerance curve than on the descent. Therefore, once the penetration of sugar through the kidney occurs, it continues though a much lower level of blood sugar concentration is present. As a result of the increased loss of sugar with water, polyuria occurs, followed by a polydipsia secondary to the dehydration that occurs. The pruritus seen in women is thought to be secondary to the irritating effect of the increased amounts of glucose in the urine.

E. *Ketosis.*—Knoop, quoted by Stadie,[25] first thought fatty acids to be oxidized by beta oxidation in which two carbon atoms were oxidized at one time with the formation of acetic acid. This continued on the long chain fatty acids until only one ketone body was left, and therefore, many acetic acid molecules were formed with only 1 ketone body. The latter was slowly oxidized only when glucose was simultaneously oxidized. If the glucose was unable to be oxidized or not present, no oxidation of the fatty acid ketone body took place and an accumulation of ketone bodies resulted. This was the "Fats burn in the flame of the carbohydrates" theory of Shaffer.[26] Hurtley in 1916 introduced the multiple alternate oxidation theory saying that oxidation of fatty acids took place at alternate carbon atoms and all at once so that many ketone bodies were produced. Stadie[25] found this to be true by quantitative experimentation and therefore opposed the Knoop theory.

He showed that fatty acids were oxidized by the peripheral tissues with or without insulin or glucose present. He further showed that diabetic tissues "call out" for energy from fat and the liver responds by utilizing fatty acids with the production of ketone bodies. These ketone bodies are utilized by the peripheral tissues. However, if the liver produces them faster than the peripheral tissues can oxidize them, then an excess occurs in the blood stream and in the urine. Stadie[25] stated the following hypothesis: "Up to a certain level all fat catabolized is completely utilized; hence, there is no ketonuria. Beyond this level all fat catabolized is not completely utilized; hence, part of the fat catabolized is excreted in the form of ketone bodies . . . The greater the carbohydrate utilized, the less the fat metabolism; that is, carbohydrate spares fats."

Soskin[21] found, by perfusion experiments, that 10-40 times as many ketone bodies were produced by the liver than by any other organ or tissue. In addition, any hepatotoxic agent decreased the production of ketone bodies, so the liver is definitely the most important organ concerned with ketogenesis. The regulation of the ketone body production will therefore depend on the integrity of the liver cell. In cases of starvation, fatty livers, and so on, there is a tendency to increase ketone production more than in the normal. Soskin mentions the endocrine involvements in the metabolic process. He says that insulin is anti-ketogenic while the thyroid, anterior pituitary, adrenal medulla, and adrenal cortical factors are all ketogenic.

Somogyi and Weichselbaum[72] found ketonuria to increase following glucose administration. If "Fats burn in the fire of carbohydrates", the ketonuria should have decreased so they concluded that carbohydrates do not exert ketolytic effects but exerted ketone sparing effects in various extrahepatic tissues and decreased ketone body utilization.

Barach[73] concluded that ketones make their appearance when inadequate carbohydrate is being metabolized and excess fat is being utilized. So, whether in health or disease, the cause for the ketosis is the same.

F. *Acidosis.*—Soskin and Levine[74] are not at all sure that the increased ketone bodies in the blood are harmful per se. But, as Gamble[75] points out, these ketone acids must be given sodium ions for neutralization purposes in order to retain a normal pH. This sodium ion is given up at the expense of the bicarbonate ion. Therefore, an increase in ketone acids is associated with a reduction of the bicarbonate content of the plasma. In addition, the rapid dehydration almost always occurring in diabetic coma is responsible for certain factors taking place which prevent renal function in the normal manner. As a result of this, more organic acid radicals are retained by the kidney, combine with sodium, and displace more bicarbonate to a very low value. Therefore, acidosis is seen to occur on an easily explainable physiological basis. Whether or not the symptoms of diabetic coma depend upon the acidosis cannot be definitely said. The coma is probably a result of the hemoconcentration of the blood stream with decreased oxygenation to the central nervous system. Soskin and Levine[74] found only evidence of cerebral capillary dilatation, perivascular edema, and acute degenerative changes in the cells of the central nervous system. They explained the changes on the basis of dehydration, hemoconcentration, and cerebral anemia.

CONCLUSION

In conculsion, I would like to repeat that diabetes mellitus is a disorder in carbohydrate metabolism secondary to insulin deficiency. All of the many manifestations of this disease may be explained on that basis. There occurs firstly a reduced rate of carbohydrate oxidation by the peripheral tissues, with a secondary, compensatory over-production of glucose from the liver which initiates the latter changes, ending in ketosis and acidosis. The importance of the liver in the normal and abnormal metabolic processes has been stressed. Therapy should be directed towards replacement of the insulin the body is lacking and replen-

ishing the glycogen depleted liver with a new supply of glycogen in an attempt to restore the normal physiologic functions.

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INFANT MORTALITY IN LOUISIANA AND UNITED STATES

The infant mortality rate per 1,000 live births was 43.0 in Louisiana in 1945 as contrasted with 46.3 in 1944. The drop in infant mortality was in line with that which has occurred throughout most of the states in the Union. The infant mortality rate in Louisiana, however, is higher than that throughout the United States as a whole, which rate is 38.3. Our rate is higher than that in the nearby Southern states: In Mississippi it is 40.6; in Alabama it is 38.3; in Arkansas 31.7. However, in Texas the rate is 48.8 which the Texans probably attribute to the high rate amongst their Mexican population. Incidentally, the state of New Mexico has the highest rate in the Union, nearly 100.8. The lowest rate in the country is in the state of Rhode Island where the mortality rate per 1,000 live births is only 28.2.

While the figure for the maternal mortality rate of each state are not given in this particular bulletin of the U. S. Public Health Service, the statement is made that the rate amongst mothers has diminished nine per cent from 1945.

The diminution in the number of infant and maternal deaths is attributed in part to the large portion of babies who are now delivered in hospitals. In 1945 the figures show that nearly 80 per cent of all deliveries were reported to have occurred in hospitals.

Comment was made concerning the infant death rate among Mexicans. It should also be noted that the infant mortality rates in the South are somewhat higher than in the North and in the West but these rates are always higher in the negro group than they are in the white population.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly this year was a truly tremendous success. The program appealed to doctors from all over the country so that actually checks came from every state in the Union asking that the writer of the check be registered for the Assembly. The unfortunate thing about this meeting was the fact that it was impossible to obtain hotel accommodations for all those who wished to

attend the meeting. As a matter of fact several hundred checks had to be returned to the senders because no rooms were to be had for them in the City of New Orleans.

The scientific program was most successful. The out of town speakers for the most part presented their subject matter clearly, succinctly and in a manner that would appeal to and impress itself upon the memory of the physician. Certainly the outside of state speakers could not have been better selected than they were. To Dr. W. H. Gillentine, president of the organization, goes much praise as he has worked assiduously in the interests of the Assembly. We feel sure that Dr. Joseph D'Antoni, the next president, will do equally well and that Dr. Waldemar R. Metz, the president-elect, will ably maintain the standard set by this organization since it has been in existence.

—o—

POSTERIOR PITUITARY EXTRACT TO MEASURE RENAL FUNCTION

Several years ago Sodeman and Engelhardt[1] devised a test for renal function which has many advantages over those that are commonly employed. Undoubtedly the most satisfactory test for renal function in most instances depends upon measuring the concentrating capactiy of the tubules. The disadvantages of previous tests have been the discomfort to the patient because of a long period of water restriction and the necessity of collecting the urine at various times during the period in which the test is going on. Sodeman and Engelhardt found that if they gave posterior pituitary extract to their patients that the results are comparable to those in which the more elaborate procedures are carried out. The only procedure necessary is to give 0.5 c.c. posterior pituitary extract subcutaneously and then collect the urine at one-half hour intervals for two hours. A recent publication by Horne and Morris[2] substantiates what Sodeman and Engelhardt observed in their original studies. Attention is called to this kidney function test and to this particular article, which is one of several that have confirmed the results obtained by the two original authors because the test is simple and dependable and permits, according to Horne and Morris, of a rapid evaluation of the concentrating powers of the kidney. The Bowman Gray School of Medicine observers furthermore found that the test in 25 patients who did not have kidney disease gave results comparable to the fluid restriction test. There was some difference in 27 patients with impaired kidney function but these changes were in no way of special significance because they were based upon the invalid assumption that one can expect the fluid restriction test always to give a constant result. "The pituitrin test determined accurately whether or not a given patient should be classified as having impaired renal function. It may be substituted for the fluid restriction test to measure the concentrating ability of the kidneys . . . ." Horne and Morris noted furthermore that this test accurately estimates the concentrating power of the kidney in patients with edema. Lastly, they concluded that the posterior pituitary test is of considerable value to the busy physician because it can be carried out in his office without any special preparation of the patient, it is finished within two hours and in the average patient causes no disturbing reaction. However, it should be borne in mind that pituitrin should not be given to the pregnant girl and potentially is dangerous to the patient who may have coronary arterial disease. The side effects of pituitrin, when given in doses of 10 U.S.P. units, are very slight. Occasionally the patient has abdominal cramps and in about 40 per cent they may have a painless stool. In no one of the patients studied by Horne and Morris were there any significant changes in the blood pressure. Parenthetically it might be observed that these authors used 1 c.c. of pituitrin and necessarily used a small calibrated urinometer.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

AGREEMENT WITH THE VETERANS ADMINISTRATION

Following is copy of contract and fee schedule now in effect between the Veterans' Administration and the Louisiana State Medical Society for medical care of veterans with service connected disabilities. Cooperation of our members in this project is earnestly requested.

CONTRACT

1. It is the purpose of the Louisiana State Medical Society to collaborate with the Veterans' Administration in a manner which will provide the best possible medical care for veterans residing in the State of Louisiana.

2. The Louisiana State Medical Society will request all of its members to participate in a statewide program whereby physicians in private practice will render medical services (examinations, treatments and counsel) in such cases as may be authorized by the Veterans' Administration.

3. The Louisiana State Medical Society will submit to the Veterans' Administration a list of members who desire to provide services for eligible veterans in home communities of such veterans.

4. The physicians so listed may be appointed as fee-designated physicians of the Veterans' Administration.

5. Such list may be augumented from time to time as additional physicians indicate a desire to participate in the program.

6. By notice in writing, a physician may at any time request that his name shall be removed from the list of fee-designated physicians.

7. Fees for medical services in authorized cases shall be paid by the Veterans' Administration to the physician rendering the service in accordance with the fee schedule, which is attached hereto and made a part of this agreement. The Louisiana State Medical Society warrants that the rates charged herein are not in excess of the rates charged other persons who are not Veterans' Administration beneficiaries for the same service. It is understood that unusually involved cases and services not scheduled will be subject to review by the Louisiana State Medical Society for recommendation to the Veterans' Administration as to the appropriate fee.

8. The Louisiana State Medical Society will assist the Veterans' Administration in establishing for examinations and treatments a list of competent specialists who meet the qualifications for spscialists of the Veterans' Administration.

9. Lists submitted by the Louisiana State Medical Society will be broken down by parishes or districts in order that the veteran for whom services are authorized may select a physician practicing in his home community.

10. The Veterans' Administration will handle administrative and clerical details in connection with the authorization of examinations or treatments and the maintenance or records; and will arrange for transportation of the veteran if necessary.

11. When authorizing examination or treatment, the Veterans' Administration will furnish to the veteran proof of such authorization and a list of fee-designated physicians in the parish or district in which the veteran is located in order that he may select his own physician for the services authorized.

12. The Veterans' Administration will review reports of examinations and services to determine their adequacy. No fees will be paid by the Veterans' Administration for reports which are not acceptable to the Veterans' Administration nor for services rendered in unauthorized cases.

13. The Louisiana State Medical Society will establish one or more boards of review composed of physicians. It shall be the duty of such board to review reports, which are deemed by the Veterans' Administration to be inadequate or which do not meet the requirements of the Veterans' Administration; to recommend, at its discretion, the disqualification of any physician from further work with the Veterans' Administration whose work is found by the board to be incomplete or unsatisfactory; to advise and assist the Veterans' Administration on other matters within the scope of this program.

14. The Louisiana State Medical Society does not propose to make any charge for any service rendered to the Veterans' Administration under this agreement.

15. This contract may be discontinued upon 30 days' notice by either participant.

Signed on behalf of the
Louisiana State Medical Society by:

RHETT McMAHON, M. D.,
President

EDWIN L. ZANDER, M. D.,
Chairman of Council

P. T. TALBOT, M. D.,
Secretary-Treasurer

Signed on behalf of the Veterans' Administration by:

R. C. KIDD, Director, Supply Service
Veterans Administration
Washington 25, D. C.

Effective date of this agreement:
December 1, 1946

FEE SCHEDULE

1. Bronchoscopy	$40.00
2. Bronchoscopy and biopsy	50.00
3. Dermatological examination	7.00
4. Electrocardiogram with interpretation	10.00
5. Esophagoscopy	40.00
6. Examination of ears, nose and throat (separately or together)	5.00
7. Special ear examination, including audiometric test, with chart	10.00
8. Special ear examination to include either caloric or Bárány test, or both, with report	10.00
9. Examination of eyes (to include either a copy of the prescription ordered or the retinoscopic correction of the refractive error, the fundus and field findings—the latter by chart in all cases of optic atrophy)	10.00
10. Examination of eyes with refraction, if mydriatic is used (to include either a copy of the prescription ordered or the retinoscopic correction of the refractive error, the fundus and field findings—the latter by chart in all cases of optic atrophy)	12.50
11. Combined examination of eyes, ears, nose and throat, with refraction (with or without mydriatic)	15.00
12. Gastroscopy	40.00
13. Genitourinary examination without cystoscopy	5.00
14. Genitourinary examination with cystoscopy	15.00
15. Genitourinary examination with cystoscopy and ureteral catheterization	25.00
16. Gynecological examination	5.00
17. Complete examination of heart, including electrocardiography	15.00
18. Physical examination of heart or lungs, or both	10.00
19. Neurological examination (complete)	5.00
20. Neuropsychiatric examination (complete)	10.00
21. Routine office examination, including treatment	3.00
22. Orthopedic examination	7.50
23. Physical examination to determine need for hospitilization	3.00
24. Complete physical examination	7.50
25. Proctoscopy or sigmoidoscopy	7.50
26. General surgical examination	5.00

SIMPLE FRACTURES

27. Carpal bone, one	35.00
28. Carpal bones, each additional	5.00
29. Clavicle	40.00
30. Coccyx removal	50.00
31. Femur	125.00
32. Femur, when suture, plating or nailing is necessary	135.00
33. Fibula or tibia, or both (including Pott's fracture)	75.00
34. Fibula or tibia, or both (including Potts fracture) when suture or plating is necessary	100.00
35. Finger, one	15.00
36. Fingers, each additional	5.00
37. Humerus	75.00
38. Humerus, when suture or plating is necessary	100.00
39. Malar bone	20.00
40. Maxilla inferior (wiring if necessary)	75.00
Double	100.00
41. Maxilla superior (wiring if necessary) or raising	75.00
42. Metacarpal bone, one	15.00
43. Metacarpal bones, each additional	5.00
44. Metatarsal bone, one	20.00
45. Metatarsal bones, each additional	5.00
46. Nasal bones	25.00
47. Patella	50.00
48. Patella, when suture or plating is necessary	75.00
49. Pelvis	100.00
50. Pelvis, when suture or plating is necessary	125.00
51. Radius or ulna, or both (including Colles' fracture)	50.00
52. Radius or ulna, or both (including Colles' fracture) when suture or plating is necessary	75.00
53. Rib, one	15.00
54. Ribs, each additional	5.00
55. Sacrum	50.00
56. Scapula	50.00
57. Skull	100.00

58. Sternum 75.00
59. Tarsal bone, one 15.00
60. Tarsal bones, each additional 5.00
61. Toe, one 15.00
62. Toes, each additional 5.00
63. Vertebra, one or more 100.00

Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

COMPOUND FRACTURES

In all compound fractures add 40 per cent additional fee.

DISLOCATIONS

64. Carpal bone, one 25.00
65. Carpal bones, each additional 5.00
66. Clavicle 35.00
67. Elbow 35.00
68. Finger, one 10.00
69. Fingers, each additional 5.00
70. Hip 60.00
71. Knee 50.00
72. Maxilla, inferior 15.00
73. Metacarpal bone, one 15.00
74. Metacarpal bones, each additional 5.00
75. Metatarsal bone, one 15.00
76. Metatarsal bones, each additional 5.00
77. Patella 40.00
78. Pelvis 75.00
79. Shoulder 40.00
80. Shoulder, recurrent or habitual 35.00
81. Tarsal bone, one 15.00
82. Tarsal bones, each additional 5.00
83. Thumb 15.00
84. Toe, one 10.00
85. Toes, each additional 5.00
86. Vertebra, one or more 100.00

AMPUTATIONS

87. Upper arm 100.00
88. Forearm 75.00
89. Finger, one 25.00
90. Fingers, each additional 10.00
91. Foot 75.00
92. Hand 75.00
93. Leg 75.00
94. Thigh 100.00
95. Toe 25.00
96. Toes, each additional 10.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

ABSCESS, INCISION AND DRAINAGE

97. Brain abscess 150.00
98. Cellulitis, incision and drainage 25.00
99. Deep abscess (including ischiorectal) .. 40.00
100. Empyema, incision and drainage, including rib resection 100.00
101. Liver abscess 150.00
102. Oral abscess (not to include dental or peridental) 15.00
103. Prostatic abscess, incision and drainage 75.00
104. Superficial abscess 5.00
105. Subphrenic abscess 150.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees except as to items numbered 99 and 104.

OPERATIONS

106. Abdominal fixation for prolapse of rectum 125.00
107. Adenectomy, cervical, inguinal, etc. (minor) 20.00
108. Adenectomy, cervical, inquinal, etc. (radical) 125.00
109. Anal fissure, operation for 75.00
110. Anastomosis, intestinal 150.00
111. Anastomosis, uretero-intestinal 150.00
112. Ankle joint, excision of 75.00
113. Apicolysis 100.00
114. Appendectomy 100.00
115. Arthroplasty, major joint 125.00
116. Biopsy 10.00
117. Bone graft (long bones) 150.00
118. Bone plate, removal of 50.00
119. Breast, resection of (simple) 75.00
120. Breast, resection of (radical) 150.00
121. Carcinoma of lower lip, excision of 50.00
122. Carcinoma of rectum, excision of 200.00
123. Carcinoma of tongue, excision of 75.00
124. Cardiospasm, dilatation for 25.00
125. Cartilage of condyle of femur, removal of 70.00
120. Breast, resection of (radical) 150.00
joint 75.00
127. Cervix, amputation of 75.00
128. Cholecystectomy 150.00
129. Cholescystotomy 100.00
130. Choledochotomy 150.00
131. Chordotomy 125.00
132. Circumcision 25.00
133. Clawfoot, operation for 75.00
134. Coccyx, excision of 50.00
135. Colostomy 100.00
136. Colporrhaphy and perineorrhaphy 100.00
137. Cystotomy, suprapubic 75.00
138. Dupuytren's contraction, operation for 100.00
139. Elbow joint, excision of 75.00
140. Epididymectomy 40.00
141. Esophagus, dilatation of by means of bougies or sounds 10.00
142. Femoral artery, ligation of 75.00
143. Fecal fistula, abdominal, operation for 150.00
144. Fistula, rectovaginal, operation for 100.00

145. Fistula, urethral, operation for
First 50.00
Second 25.00
146. Fistula, vesicovaginal, operation for 75.00
147. Fistula-in-ano, operation for 75.00
148. Fulguration of tumor of bladder
Closed 50.00
Open 75.00
149. Fulguration of tumor, superficial 10.00
150. Gasserian ganglion, excision of 150.00
151. Gastrectomy (partial) 150.00
152. Gastroenterostomy 150.00
153. Hallux valgus, operation for 75.00
154. Hallux valgus, bilateral, operation for 75.00
155. Hammer toe, operation for 25.00
156. Heart, operation on 150.00
157. Hemorrhoidectomy 75.00
158. Herniotomy, diaphragmatic 150.00
159. Herniotomy, ventral, $100.00, inguinal, $75.00, or femoral 75.00
160. Herniotomy, ventral, $100.00, inguinal, or femoral (bilateral) 125.00
161. Hip joint, excision of 150.00
162. Hydrocele, aspiration of 5.00
163. Hydrocele, operation for 50.00
164. Hysterectomy, abdominal or vaginal (including removal of adnexa, if indicated) 150.00
165. Ingrown toenail, excision of 15.00
166. Intestinal obstruction, operation for 100.00
167. Knee joint, excision of 100.00
168. Laminectomy 150.00
169. Laparotomy and drainage, general peritonitis 125.00
170. Litholapaxy 75.00
171. Lobectomy 150.00
172. Meckel's diverticulum, excision of 125.00
dependent on site ..... $40.00—100.00
173. Nephrectomy or nephrotomy 150.00
174. Nephropexy 150.00
175. Nerve, suture of 100.00
176. Supraorbital nerve, injection of 10.00
177. Neuroma, resection of 75.00
178. Orchidectomy 75.00
179. Osteomyelitis, operation for—
dependent on site ..... $40.00—100.00
180. Ovariotomy 100.00
181. Papilloma of bladder, operation for 100.00
182. Paracentesis of abdomen 15.00
183. Paracentesis of pericardium 25.00
184. Paracentesis of thorax 25.00
185. Perineum, repair of 75.00
186. Phrenic nerve operation 75.00
187. Pneumolysis, extrapleural or intrapleural 100.00
188. Pneumonectomy 200.00
189. Pneumonotomy, cautery 100.00
190. Pneumoperitoneum, first induction 25.00
191. Pneumoperitoneum, refills 10.00
192. Artificial pneumothorax, first induction 25.00
193. Artificial pneumothorax, refills 10.00
194. Prostatectomy, perineal 150.00
195. Prostatectomy, suprapubic (one or two stages) 150.00
196. Prostatic resection, transurethal 125.00
197. Cisterna puncture, including local anesthetic and obtaining fluid 50.00
198. Lumbar puncture, including local anesthetic and obtaining fluid 10.00
199. Pyelotomy, with removal of calculus 150.00
200. Pyloroplasty 125.00
201. Salpingectomy 150.00
202. Scaleniotomy 50.00
203. Sequestrum, removal of (deep) depending on size and location 150.00
204. Sequestrum, removal of (superficial) 25.00
205. Shoulder joint, excision of 125.00
206. Skull, decompression of 150.00
207. Fixation of spine, operation for (Albee or Hibbs') 150.00
208. Splenectomy 125.00
209. Stricture of rectum, operation for 50.00
210. Sympathectomy, cervical 175.00
211. Sympathectomy, periarterial 100.00
212. Tenorrhaphy, one 40.00
213. Tenorrhaphy, each additional 15.00
214. Tenotomy 35.00
215. Thoracotomy 100.00
216. Thoracoplasty, each stage 125.00
217. Thyroid artery, ligation of 75.00
218. Thyroidectomy 150.00
219. Torticollis, operation for 75.00
220. Tumor, abdominal, removal of 125.00
221. Tumor of brain, operation for 200.00
222. Tumor, gastrointestinal tract, resection of, including intestinal anastomosis 200.00
223. Tumor or cyst, removal of, depending on site ..... $25.00—50.00
224. Tumor or cyst, removal of 10.00
225. Ulcer, gastric or duodenal, operation for 150.00
226. Ureteral stone, removal of 125.00
227. Urethral stricture, dilatation of 5.00
228. Urethrotomy, external 40.00
229. Urethrotomy, internal 40.00
230. Prolapsus uteri, operation for, including perineal repair 150.00
231. Uterine displacement, abdominal, operation for 125.00
232. Uterus, dilatation and curretage of 50.00
233. Varicocele, operation for 50.00
234. Varicose veins, injection treatment, each injection 5.00
235. Varicose veins, one leg, operation for 75.00
236. Varicose veins, both legs, operation for 125.00
237. Venesection 15.00
238. Whitehead's operation 75.00

239. Wrist joint, excision of 75.00
Note—these amounts include 30 days' postoperative care, exclusive of hospital charges, anesthetic and x-ray fees.

SURGICAL CARE OF TRAUMATIC WOUNDS

240. Incised, depending on size and location ..... $5.00—50.00
241. Lacerated, depending on size and location ..... $5.00—50.00
242. Punctured, depending on size and location ..... $5.00—50.00
Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

ANESTHESIA

243. Avertin anesthesia 10.00
244. General anesthesia
(a) By visiting physician 15.00
(b) By intern or nurse 5.00
(In case of gas anesthesia, an additional allowance of $5.00 may be authorized for the gas used.)
245. Local anesthesia—No fee will be allowed for local anesthesia in cases which require a local anesthetic for examination, treatment, or surgical operation.
246. Rectal anesthesia 10.00
247. Spinal anesthesia, including anesthetic 20.00

EYE OPERATIONS

248. Cataract, needling operation for 50.00
249. Cataract, operation for 100.00
250. Chalazion, operation for 10.00
251. Corneal ulcer, cauterization of 10.00
252. Extensive peripheral ulcer, cauterization of 20.00
253. Ectropion, operation for 50.00
254. Entropion, operation for 50.00
255. Enucleation of eye 75.00
256. Foreign body, removal from conjunctiva (dissection) 15.00
257. Foreign body, removal from conjunctiva (magnet) 10.00
258. Foreign body, removal from cornea (dissection) 25.00
259. Foreign body, removal from cornea (magnet) 20.00
260. Foreign body, removal from eyeball (deep) ..... $50.00—75.00
261. Grattage of lids for trachoma 10.00
262. Hordeolum, operation for 5.00
263. Iridectomy 75.00
264. Lacrymal duct, dilatation of 10.00
265. Lacrymal sac, excision of 50.00
266. Pterygium, operation for 50.00
267. Ptosis, skin and tarsal resection, operation for 75.00
268. Strabismus, operation for 100.00
Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

EAR OPERATIONS

269. Mastoid, acute, operation for 100.00
270. Mastoid, radical, operation for 150.00
271. Ossiculectomy 75.00
272. Paracentesis 10.00
273. Polypus, removal of 25.00
274. Lateral sinus, drainage of 150.00
Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

NOSE AND THROAT OPERATIONS

275. Adenoidectomy 25.00
276. Antrum, intranasal, drainage of 25.00
277. Antrum, radical, operation for 100.00
278. Cleft palate, operation for, complete closure 150.00
279. Harelip, operation for 75.00
280. Intubation 35.00
281. Laryngectomy 150.00
282. Larynx, cauterization of 10.00
283. Tumor of larynx, removal of 100.00
284. Nasal polypus, removal of 25.00
285. Nasal septum, submucous, resection of 75.00
286. Pharyngeal abscess, operation for 20.00
287. Accessory nasal sinuses, irrigation of 10.00
288. Ethmoid sinus, radical, operation for .. 75.00
289. Frontal sinus, intranasal, drainage of 50.00
290. Frontal sinus, radical, operation for 100.00
291. Sphenoid sinus, drainage of 50.00
292. Tonsillar abscess, operation for 20.00
293. Tonsillectomy 35.00
294. Tonsillectomy and adenoidectomy 45.00
295. Tracheotomy 75.00
296. Turbinate bone, galvano-cauterization of 20.00
297. Turbinectomy 25.00
Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

X-RAY WITH INTERPRETATION

298. Abdomen, flat plate 10.00
299. Ankle joint, anteroposterior and lateral views 7.50
300. Arm, humerus, anteroposterior and lateral views 10.00
301. Bladder, with injection, anteroposterior view 10.00
302. Chest, for pulmonary, cardiac or rib fracture diagnosis, plain 10.00
303. Chest, for pulmonary, cardiac or rib fracture diagnosis, stereoscopic 15.00
304. Clavicle, anteroposterior view 7.50

305. Elbow, anteroposterior and lateral views 7.50
306. Fluoroscopy, when required, without film 5.00
307. Foot, anteroposterior and lateral views 7.50
308. Forearm, radius and ulna, anteroposterior and lateral views..... 7.50
309. Foreign body in eye, location of (the fragment charted in three planes and its dimensions ascertained by the method of Sweet or equivalent).. 25.00
310. Gall bladder, Graham technic, including cost of dye..... 15.00
311. Gastrointestinal tract, complete x-ray study including fluoroscopy..... 35.00
312. Hand, anteroposterior and lateral views 5.00
313. Hip joint, anteroposterior view, plain 10.00
314. Hip joint, anteroposterior view, stereoscopic 15.00
315. Intestine, barium clysma, 14 by 17 films for position and outline..... 10.00
316. Jaw, upper and lower..... 7.50
317. Kidneys, right and left for comparison 10.00
318. Knee joint, anteroposterior and lateral views 7.50
319. Leg, tibia and fibula, anteroposterior and lateral views 7.50
320. Lipiodol injection for bronchiectasis, etc., including roentgenograms..... 25.00
321. Pelvis, anteroposterior view, plain..... 10.00
322. Pelvis, anteroposterior view, stereoscopic 15.00
323. Pyelography, using uroselectan or similar preparation (including cost of drug) 15.00
324. Pyelography, retrograde 25.00
325. Scapula 7.50
326. Shoulder joint, anteroposterior view, plain 7.50
327. Shoulder joint, anteroposterior view, stereoscopic 10.00
328. Sinuses, frontal and ethmoid, anteroposterior and lateral views..... 10.00
329. Sinuses, mastoid, right and left sides for comparison 10.00
330. Sinuses, maxillary, anteroposterior and lateral views..... 10.00
331. Sinuses, frontal, ethmoid and maxillary, anteroposterior and lateral views 15.00
332. Skull, anteroposterior and lateral views, plain 10.00
333. Skull, anteroposterior and lateral views, stereoscopic 15.00
334. Spine, cervical, anteroposterior and lateral views 10.00
335. Spine, dorsal, anteroposterior and lateral views 12.50
336. Spine, lumbosacral, with coccyx, anteroposterior and lateral views..... 12.50
337. Spine, entire 22.50
338. Stomach, barium or bismuth meal, 14 by 17 film; after ingestion, four 8 by 10 films for detection of duodenal cap; total of five films including fluoroscopy 25.00
339. Teeth, single (up to and including 7 films) each 1.00
340. Teeth, series (7 films to and including full mouth) 7.00
341. Thigh, femur, anteroposterior and lateral views 7.50
342. Wrist, anteroposterior and lateral views 5.00

INTERPRETATION OF ROENTGENOGRAMS

343. Bones and joints, plain anteroposterior and lateral views..... 5.00
344. Chest for pulmonary diagnosis, plain or stereoscopic 5.00
345. Gastrointestinal series 5.00
346. Genitourinary tract 5.00
347. Kidney films 5.00
348. Skull, following ventriculography or encephalography 5.00
349. X-ray therapy, deep, per treatment .....$10.00—25.00 (Maximum expenditure allowed not to exceed $100.00; where additional treatments are necessary, special authority must be obtained from Central Office.)
350. X-ray therapy, superficial..... 5.00

MISCELLANEOUS

351. Blood transfusion 35.00
352. Amount allowed for blood furnished, when not donated, per 100 c.c..... 5.00
353. Application of plaster cast, chest (including material) 15.00
354. Application of plaster cast, thighs and hips (including material)..... 25.00
355. Application of plaster cast, thigh and leg (including material)..... 10.00
356. Application of plaster cast, torso (including material) 25.00
357. Application of plaster cast, torso and hips (including material)..... 30.00
358. Application of plaster cast, torso, entire body, (chest to feet, including material) 50.00
359. Application of plaster cast for disease or injury of vertebrae (including material) 15.00

Office visit $2.00
Day calls to home or hospital..... 3.00

Night calls to home or hospital..... 5.00
(For all calls within city limits)
Mileage (one way within zone, beginning at city limits, per mile)..... .75

It will be the duty of the Louisiana Medical Society to see that the state is zoned whereby the nearest physician in rural districts must be called in case of emergency. The veteran calling a more distant physician will pay the additional mileage.

CLINICAL LABORATORY EXAMINATIONS

BACTERIOLOGICAL EXAMINATIONS

1. Cultural examination for fungi..... $5.00
2. Microscopic examination for fungi..... 2.00
3. Pneumococcus typing 5.00
4. Pus or exudate (smear)..... 1.00
5. Pus or exudate, cultural exam, classification 5.00
6. T. Pallidum (dark field)..... 5.00
7. Throat culture, classification of organism 5.00
8. Throat smear 1.00

BLOOD

9. Agglutination test for typhoid, paratyphoid, or undulant fever..... 2.00
10. Bleeding time 1.00
11. Blood calcium 3.00
12. Blood chlorides 3.00
13. Blood culture, including classification 5.00
14. Blood platelet count..... 1.00
15. Blood smear for malaria..... 2.00
16. Blood typing (grouping)..... 1.00
17. Carbon dioxide combining power of blood plasma 5.00
18. Chemical examination of blood, complete, including creatinin, dextrose, urea, nitrogen (or non-protein N) and uric acid..... 15.00
19. Cholesterol 3.00
20. Coagulation time 1.00
21. Complement fixation test — gonococcus infection 3.00
22. Complement fixation test for syphilis.. 3.00
23. Complement fixation test for tuberculosis 3.00
24. Creatinin 3.00
25. Dextrose 3.00
26. Total erythrocyte count..... 2.00
27. Fragility test for erythrocytes..... 5.00
28. Hemoglobin estimation 2.00
29. Hydrogen ion concentration..... 5.00
30. Differential leukocyte count..... 2.00
31. Total leukocyte count..... 2.00
32. Complete blood count, including total counts 5.00
33. Non-protein nitrogen 3.00
34. Non-protein nitrogen 3.00
35. Blood phosphorus 2.00
36. Precipitation test for syphilis..... 2.00
37. Reticulocyte count 2.00
38. Sedimentation rate 2.00
39. Estimation of sugar tolerance..... 10.00
40. Urea nitrogen 3.00
41. Uric acid 3.00
42. Van den Bergh blood test for icterus.. 2.00
43. Volume index 3.00

FECES

44. Cultural exam. of feces for causative micro-organism (classification of bacterium) 7.50
45. Fat in feces..... 1.00
46. Parasites and ova..... 5.00

PATHOLOGICAL EXAMINATIONS

47. Autopsy, complete, with report, including histological examination..... 50.00
48. Tissue examination, with report..... 5.00

SKIN TESTS

49. Protein sensitization tests (series) including allergens, for purpose of establishing causative factor..... 25.00
50. Tuberculin 3.00

SPINAL FLUID

51. Examination of spinal fluid for causative organism (smear)..... 3.00
52. Cell count 2.00
53. Colloidal gold reaction..... 5.00
54. Complement fixation test for syphilis 3.00
55. Cultural exam. of spinal fluid, including the classification of causative microorganisms 5.00
56. Globulin test 1.00
57. Complete exam. of spinal fluid, including complement fixation test, colloidal gold, globulin test, and cell count 10.00
58. Precipitation test for syphilis..... 3.00

SPUTUM

50. Tubercle bacillus (plain smear)..... 3.00
60. Tubercle bacillus (concentration method) 5.00
61. Exam. of duodenal content for pancreatic ferments 3.00
62. Exam. of gastric content for acidity, by histamine 7.50
63. Exam. of gastric content for pepsin.... 3.00
64. Routine chemical (including test meal with withdrawal of stomach contents) 10.00

URINE

65. Chemical examination, routine..... 1.00
66. Chemical and microscopical examination 2.00
67. Chlorides 3.00
68. Creatinin 3.00
69. Cultural exam., including classifica-

tion of microorganism	5.00
70. Hydrogen ion concentration	1.00
71. Mosenthal test	2.00
72. Total nitrogen	3.00
73. Renal function test (including phenosulphonephthalein)	3.00
74. Tubercle bacilli	3.00
75. Urea nitrogen	3.00
76. Uric acid	3.00
77. Urobilin	1.00
MISCELLANEOUS EXAMINATIONS	
78. Animal inoculation for diagnosis, with report of autopsy	10.00
79. Preparation of autogenous vaccine	10.00
80. Determination of basal metabolic rate	5.00
MISCELLANEOUS	
81. Blood transfusion (administration only, without venesection)	5.00
82. Non-surgical drainage of gallbladder	10.00
83. Electrocardiograms, interpretation of	5.00
84. Hypodermoclysis	3.00
85. Injection of alcohol, trigeminal nerve	25.00
86. Intravenous injection, exclusive of cost of drug	3.00

LOUISIANA STATE PHARMACEUTICAL ASSOCIATION

The Veterans' Administration-Pharmacy Service Agreement with the Louisiana State Pharmaceutical Association has been approved and is now in effect. Information as to participating member pharmacies may be secured by contacting the Veterans' Advisory Bureau, L.S.P.A., Sta. B.—P. O. Box 5317, New Orleans 15.

There are no restrictions upon physicians as to medical agents which may be prescribed but the term "medical requisite" in the agreement is defined as including only the following items: Insulin syringe and two needles; two hypodermic (insulin type) needles; atomizer; nebulizer; hot water bottle; fountain syringe; combination hot water bottle and syringe; ice bag; ice cap; urinal; bed pan; enema can; feeding tube; ear and ulcer syringe. These items may be prescribed only in the case of an emergency and each prescription must be limited to a single item. Pharmacists can not be reimbursed for refilling any prescription and for this reason it will be necessary for the doctors of the state to write new prescriptions whenever additional drugs are indicated or a new medical requisite is required.

For additional data contact the Veterans' Advisory Bureau, L.S.P.A. at the above mentioned address.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

March 10 Scientific meeting, Orleans Parish Medical Society, 8 p. m.

March 12 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

March 14 Executive Committee, Hotel Dieu, 8 p. m.

March 17 Hotel Dieu Staff, 8 p. m.

March 18 I. C. R. R. Hospital, 12:30 p. m.
Charity Hospital Medical Staff, 8 p. m.

March 19 The Orleans Society of X-ray Technicians, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.

March 20 Clinico-pathologic Conference, Touro Infirmary, 12 noon.

March 21 Lakeshore Hospital, 8 p. m.

March 25 Baptist Hospital Staff, 8 p. m.

March 26 French Hospital Staff, 8 p. m.

March 27 DePaul Sanitarium Staff, 8 p. m.

March 28 L. S. Faculty Club, 8 p. m.
New Orleans Dispensary for Women and Children Staff, 8 p. m.

April 1 Orleans Parish Radiological Society, 7:30 p. m.
Eye, Ear, Nose and Throat Staff, 8 p. m.

April 2 Mercy Hospital Staff, 8 p. m.

April 3 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Executive Committee, Baptist Hospital, 8 p. m.

April 4 Ochsner Clinic Staff, 8 p. m.

April 7 Board of Directors, Orleans Parish Medical Society.

April 9 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

Every Thursday—Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.
N. J. Tessitore, M. D., Secretary.

NEWS ITEMS

Dr. Jules Myron Davidson was elected national Vice-Grand Council of Phi Delta Epsilon, National Medical Fraternity, at their recent meeting in York City.

Dr. F. E. Lamothe was installed as Potentate of Jerusalem Temple at ceremonies held Thursday, January 9.

Dr. A. Mattes was installed as chief rabban.

Dr. I. L. Robbins spoke on the life of Maimonides, twelfth century theologist and philosopher, at a meeting of the Tulane History of Medicine Society held January 10.

Dr. Alton Ochsner recently attended a meeting of the subcommittee on thoracic surgery of the National Research Council in Washington.

Dr. Ochsner also recently addressed the American Cancer Society in New York, and the Academy of Medicine in Buffalo.

Dr. Daniel N. Silverman attended the first meeting of the 1947 Jefferson County Medical Society in Birmingham, January 20-22. Dr. Silverman spoke on the Recognition of the Usual and Unusual Types of Dysentery and Their Treatments. While in Birmingham Dr. Silverman also addressed the faculty and student body of the Alabama Medical School.

Dr. George Battalora was recently elected President of the Mercy Hospital Staff.

Dr. Battalora succeeds Dr. E. J. Richard, who was named to the executive committee.

Other officers elected were: Dr. F. F. Boyce, vice-president; Dr. Louis Monte, secretary; Dr. Edmond Mickal, treasurer; and Dr. E. L. Zander, member of the executive committee.

Dr. Edmond Souchon was recently installed as President of the Hotel Dieu staff; he succeeds Dr. Lucien A. Fortier.

Other officers installed were: Dr. O. C. Cassegrain, vice-president; and Dr. J. O. Weilbaecher, Jr., secretary-treasurer.

Dr. H. Randolph Unsworth was recently elected as President of the DePaul Sanitarium staff; he succeeds Dr. C. S. Holbrook.

Other officers elected were: Dr. Lucy Scott Hill, vice-president and Dr. Louis J. Dubos, secretary-treasurer.

SCIENTIFIC SESSIONS

The result of the recent survey, concerning the type of scientific programs the members prefer, is as follows:

One hundred, ninety-one members replied.

40 mixed papers only.
37 symposia only.
21 symposia and mixed papers.
18 specialty only.
17 symposia, and schools, hospitals, etc.
14 symposia, mixed papers, and schools, hospitals, etc.
12 symposia and specialty.
8 specialty, and schools, hospitals, etc.
6 symposia, specialty, and schools, hospitals, etc.
5 mixed papers and schools, hospitals, etc.
4 medical school, hospitals, etc. only.
3 all four types of programs.
2 specialty and mixed papers.
1 mixed papers and hospitals only
1 doesn't matter.
2 contained no signature and were not included in the count.
1 in addition to making a selection of the types of meetings, suggested more social affairs.

The Scientific Essays Committee is, at present, preparing programs for the year, if you have not returned your card indicating the type of programs you prefer, or informing the Committee as to whether you wish to participate in the programs, it is not too late to do so. The Committee, in preparing the programs, will make the schedule very flexible.

CHANGES IN ADDRESS

Dr. Peter Everett, 502 Pere Marquette Bldg.

Dr. Prudence E. Prouet, 1007 Pere Marquette Bldg.

Dr. Curtis W. Caine, U. S. Naval Hospital, Portsmouth, Virginia.

Dr. J. Wm. Rosenthal, U. S. Veterans Hospital, Fayetteville, Arkansas.

Dr. George E. Scheer, U. S. Quarantine Station, Rosebank, Staten Island 5, New York.

CHANGES IN MEMBERSHIP ROLLS

Dr. W. J. Gilbert, an active member, and Dr. Carey C. Womble, an associate member, were elected to inactive membership.

Dr. Paul M. Goldfarb transferred from intern to active membership.

Drs. Milton R. Cohen, Ralph H. Heeren, Joseph D. Lea, and Roland F. Phillips, all active members, were removed from the rolls because of removal from New Orleans.

HOSPITAL NEWS

The following New Orleans hospitals recently received from the American College of Surgeons, certificates of approval for complying satisfactorily with fundamental requirements for caring for the sick and injured:

Charity, DePaul, Eye Ear Nose and Throat,

Flint-Goodridge, Hotel Dieu, Illinois Central, Mercy, New Orleans Hospital Dispensary for Women and Children, Southern Baptist and Touro Infirmary.

NEW ORLEANS MEDICAL FOUNDATION

On January 14 the New Orleans Medical Foundation held its first annual meeting.

The secretary, Dr. L. Sidney Charbonnet, Jr., reported on the activities of the year—the main endeavor being the establishment of the Lakeshore Hospital; he reported that 2,500 patients had been treated in the Hospital between the dates of June 1, 1946 and January 1, 1947, the census having averaged 78 patients. Dr. Charbonnet further pointed out the necessity of complete cooperation of the membership to increase the census and thus meet the expenses of operation of the Hospital, which has maintained very high standards of service.

The treasurer, Dr. Pascal Danna, reported that $125,000 had been expended in the renovation, equipping and organization of Lakeshore Hospital, and that the operating cost averaged $35,000 a month.

Dr. W. P. Gardiner, director of the Hospital, stressed the excellent services that are available for both patients and doctors, and invited all the doctors of the Society to utilize the Hospital facilities which now include adequate beds on all services.

Election of officers was carried out at this meeting—the following were re-elected for the coming year: Dr. Charles B. Odom, president; Dr. J. O. Weilbaecher, Jr., first vice-president; Dr. C. Barrett Kennedy, second vice-president; Dr. L. Sidney Charbonnet, Jr., secretary; Dr. Pascal L. Danna, treasurer; to the executive committee (members-at-large), Drs. George M. Haik, Warren L. Rosen, Chester S. Fresh, Gilbert C. Tomskey, and Alfred B. Longacre.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THIRD DISTRICT MEDICAL SOCIETY

At a recent meeting of the Third District Medical Society, reactivated on 15 January, since its inactivation during the war, there was the largest representation proportionately present at any time in the district's history.

Dr. F. H. Davis, of Lafayette, was elected president, Dr. L. A. Morough, vice-president, Drs. L. Villien and H. M. Flory, delegate and alternate, respectively, to the state convention. Dr. O. E. Dalton was elected secretary-treasurer.

Dr. W. P. D. Tilly gave a very comprehensive and interesting dissertation on medical society organization in this area. Dr. Rhett McMahon, president of the Louisiana State Medical Society, gave a brief talk on unity of the medical profession, socialized medicine, and the future of our Louisiana Physicians Service, Inc. It was unfortunate that Dr. McMahon's talk (especially the extemporaneous parts were not reported verbatim). Dr. Talbot was kind enough to stimulate the meeting with his presence. Mr. Frank Lais, the executive secretary of the Louisiana Physicians Service, Inc., gave a brief explanation of what could be expected from it.

Dr. C. C. DeGravelles, of New Iberia, made a motion which was seconded, and carried, that the next meeting be held in New Iberia.

Dr. L. J. Sonnier acted as secretary.

Orien E. Dalton, M. D.,
Secretary-Treasurer.

WEBSTER PARISH MEDICAL SOCIETY

The Webster Parish Medical Society met at 7:00 p. m., January 27, 1947 at the Imperial Hotel in Minden, La. Supper was served to 50 members, guests, and their wives. Dr. Alton Ochsner of New Orleans spoke on Frequent Errors in the Early Diagnosis and Treatment of Cancer.

E. S. Rogers, M. D.,
Secretary.

THE IBERIA PARISH MEDICAL SOCIETY

The Iberia Parish Medical Society met on January 23, 1947 at a supper-meeting in New Iberia.

Definite steps were taken to hold meetings quarterly and adopt a scientific program to be presented by the members, talks of five minutes with two minutes' discussion periods. Presiding was Dr. Leon Slipakoff, president, and Dr. I. W. Gajan, secretary-treasurer. Members present were Drs. Tilly, DeGravelles, Phillips, Dupuy, Ed L. Landry, Flory, and Dalton. Officers elected for 1947 are: President, Dr. O. E. Dalton; Vice-President, Dr. C. W. Phillips; Secretary-Treasurer, Dr. I. W. Gajan, Jr.; Delegate, Dr. E. L. Landry; Alternate, Dr. Leon Slipakoff.

I. W. Gajon, Jr., M. D.
Secretary-Treasurer.

RAPIDES PARISH MEDICAL SOCIETY

The regular meeting of the Rapides Parish Medical Society was held at the Hotel Bentley on February 3. The meeting was preceded by a dinner after which the scientific program was arranged for by the members of the staff of the Veterans Hospital at Alexandria.

TOURO INFIRMARY

The regular meeting of the Medical Staff of Touro Infirmary was held on Wednesday, February 12 at 8 p. m. Miss Ruth Corpron gave a statistical study of parasitology in the hospital. Dr. Michael DeBakey reported on a case of esophagogastrectomy and Dr. J. K. Howles spoke about lymphoblastoma of the skin.

THE WILLIS-KNIGHTON CLINIC

The Willis-Knighton Clinic announces the following additions to its staff: Dr. Frank L. Bryant, formerly of Minneapolis, who was chief of ophthalmology and otolaryngology in the Oak Ridge, Tennessee, Hospital during the war. Dr. B. H. Young, who was in the Medical Corps of the U. S. Navy, and who is a graduate of Tulane University, has become affiliated with the department of internal medicine. Dr. Herbert H. Vaughan, Jr., formerly associated with the hospital and instructor in the University of Michigan School of Medicine, is now connected with the department of surgery.

CONGRESS ON OBSTETRICS AND GYNECOLOGY

The third American Congress on Obstetrics and Gynecology will be held in St. Louis, September 8-12, 1947. Dr. Woodard Beacham is on the committee in charge of the technical exhibits.

Dr. Fred L. Adair is General Chairman of the Congress. Papers will be presented by leading obstetricians, gynecologists, public health officials, hospital administrators and others. There will be round table discussions and group meetings as well.

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER

A most elaborate and excellent program has been arranged for the meeting of the American Association for the Study of Goiter which will be held at the Biltmore Hotel, Atlantic City, April 2-5, 1947.

THE AMERICAN SOCIETY FOR THE STUDY OF STERILITY

The third annual convention of this organization will be held at the Hotel Strand, Atlantic City, June 7-8, immediately preceding the A. M. A. Convention.

SOUTHERN REGIONAL CONFERENCE ON MEDICAL SERVICE AND PUBLIC RELATIONS

The Council on Medical Service of the American Medical Association has arranged a Southern Regional Conference in cooperation with the Council on Medical Service and Public Relations of the Louisiana State Medical Society. The conference will be held in New Orleans on Saturday, March 15, in the University Room, Roosevelt Hotel. The conference is a one day meeting and registration will start at 9 a. m.

The States of Alabama, Arkansas, Louisiana, Mississippi, Oklahoma and Texas will participate in the conference.

The conference is open to all members of the State Society and members expecting to attend the meeting are advised to communicate with the office advising them of their attendance.

Dr. A. V. Friedrichs is chairman of the arrangements.

Numerous prominent national figures will appear on the program.

9:00 a. m.	Registration.
9:30 a. m.	Word of Welcome—G. C. Anderson, M. D. and H. Ashton Thomas, M. D.
9:45 a. m.	Purposes of Meeting—James R. McVay, M. D.
10:00 a. m.	Liaison between A. M. A. and the State Medical Societies—George F. Lull, M. D., Secretary and General Manager, American Medical Association.
10:30 a. m.	The Washington Legislative Picture—Joseph S. Lawrence, M. D., Director, Washington Office of the Council on Medical Service.
11:00 a. m.	Rural Medical Care—J. Paul Jones, M. D., Camden, Ala. Reports of the States.

12:15 a. m. Luncheon

2:00 p. m. Public Relations Roundtable Discussion—Moderator — Mr. Henry S. Johnson, Director of Public Relations, Medical Society of Virginia.

NEWS ITEMS

The Stars and Bars Chapter of Alpha Omega Alpha held its annual banquet at the St. Charles Hotel on Thursday, February 20. Dr. Dilworth W. Woolley of the Rockefeller Institute for Medical Research addressed the group. On Friday, February 21 the annual oration was delivered by Dr. Woolley in the Hutchinson Memorial on the topic "Development and Uses of Antivitamins."

Dr. George E. Burch, Associate Professor of Medicine at Tulane University, delivered the annual Alpha Omega Alpha address to the members of the chapter at Emory University on February 20. The title of his address was "Studies with Radiosodium in Congestive Heart Failure."

Drs. Edgar Hull, Edward deS. Matthews and Wm. H. Gillentine attended the meeting of the Southeastern Clinical Club in Birmingham on February 15 at which time Dr. Gillentine was elected president of the organization.

Dr. Wm. H. Bloch, of New Orleans, has been honored with life membership in the Alumni Association of the University of Maryland, located at Baltimore. He has been an active member of this association for fifty years.

Colonel Joseph H. McNinch, M. C., has been designated Commandant of the Army Medical Library by The Surgeon General, succeeding Colonel Leon L. Gardner.

Major Douglas Lindsey, who obtained his Majority on October 4, 1946, is at present Deputy Director of the Department of Tactics and Technique at the Medical Field Service School, Fort Sam Houston, Texas.

The American Association of Obstetricians, Gynecologists and Abdominal Surgeons announces a foundation prize contest.

For further information write Dr. James R. Bloss, Secretary, 418 Eleventh Street, Huntington 1, West Virginia.

DOCTOR WANTED

Rating Specialist, Medical, is needed by the Shreveport Regional Office of the Veterans Administration. For details contact office of the State Society or Mr. Thomas Hurt, Shreveport Regional Office.

PHYSICIANS IN CIVILIAN PRACTICE DURING WAR URGED TO REPLY ON NEW QUESTIONNAIRE

Parallel with the advance of scientific warfare there has been a rapid advance in scientific medicine in the twentieth century. The huge strides made in our ability to destroy life and health in the recent war, however, have left many problems which remain to be solved. One of the most critical problems, as recognized by the House of Delegates of the American Medical Association in its December meeting is that of providing care for the civilian population in the event of another national emergency.

The medical profession is wisely assuming its obligation to the American people to be prepared with facts and recommendations for any such future emergency. As a first step in this preparation, 5,000 questionnaires have gone out to a list of physicians, selected at random, who passed the recent war years in civilian practice.

Every physician receiving one of these questionnaires has a professional and humanitarian duty to perform in completing it and returning it promptly. It is preferred that each sign his name, but if anyone wishes to remain anonymous he may do so. The important thing is to get as broad a representation of facts, experiences, and opinions as possible.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana in the week ending January 11 showed the following diseases appearing in numbers greater than ten: 58 cases of pulmonary tuberculosis, 26 of influenza, 20 of septic sore throat and 14 of typhus fever. There were two cases of poliomyelitis listed this week. Of the 14 cases of typhus fever, seven of them were reported from Orleans Parish.

For the week which closed January 18, the indications are that there was a widespread epidemic of mumps as 335 cases were reported this week, followed in order of frequency by chickenpox in 69 instances, 35 of influenza, and 25 of pulmonary tuberculosis. There were no cases of poliomyelitis reported this particular week.

In the week ending January 25, pulmonary tuberculosis led all other reportable diseases with 32 cases, followed by 29 of influenza and 14 of chickenpox. Two cases of poliomyelitis were listed this week and two of malaria.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending December 31, 1946

	Total This Month	Total Previous Months	Total to Date 1946
Chancroid	29	595	624

Gonorrhea	1,034	16,995	18,029
Granuloma inguinale	20	280	300
Lymphopathia venereum	7	99	106
Syphilis	900	18,250	19,150

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending January 4 there were 159 deaths in the City as contrasted with 172 the previous week. These deaths were divided 104 white, 55 negro and of the group 22 were children under one year of age.

For the following week which closed January 11 almost exactly the same number of people died in the city as had died the previous week, namely 156. Of these 100 were white, 56 were non-white and 12 were children under one year of age.

The following week which terminated January 18, showed a remarkable reduction in the number of deaths in the city, in that 113 of the citizens of the city expired this particular week. Seventy-four of these were white, 39 were negro and only 9 were children under one year of age.

The following week which ended January 25, as would be expected, showed a sharp increase in the number of deaths in the city. This week there were 165 deaths divided 108 white, 57 negro and seven infants.

There have been marked fluctuations in the deaths in the city. In the week which began the month of February there was a drop of 124 in the number of deaths, divided 76 white, 33 colored, plus 15 infant deaths.

WOMAN'S AUXILIARY

Mrs. Rhodes J. Spedale, public relations chairman of the Woman's Auxiliary to the Louisiana State Medical society announces the beginning of a series of radio health broadcasts entitled "Melody of Life" arranged through the co-operation of radio station WLCS of Baton Rouge.

The first broadcast was scheduled to go on the air at 4 p. m. Tuesday, January 21 and the program will be heard at that same time weekly for 13 weeks.

The broadcasts will be from transcription prepared by the American Medical Association, with the recording and production facilities available in Chicago, where many of the well-known network radio broadcasts originate. Materials are from the library and the publications of the American Medical Association. Numerous eminent physicians, both in medical practice and public health work, have been brought before the microphone to be interviewed for these "platters". All scripts are professionally prepared by experienced radio writers, and the voices, other than those of the doctors, are those of established radio personalities, among whom are included Harriet Hester, formerly of the staff of WLS, Chicago, and June Merrill women's feature announcer for WJJD, Chicago. Harriet Hester and William J. Murphy, veteran writer of medical scripts, have written the continuities.

The series is under the general direction of Dr. W. W. Bauer, director of the Bureau of Health Education of the American Medical Association, who appears on many of the programs, and his associate in the bureau, Dr. William W. Bolton, whose voice also is heard. Dr. Bauer is general director of the radio work of the American Medical association.

Included in these transcriptions will be the lives of the following men: Mendelssohn, Handel, Schumann, Beethoven, Chopin, Mozart, Brahms, Foster, Moussorgsky, Tchaikowski, Dvorak, Borodin and Bach.

ST. TAMMANY PARISH AUXILIARY

Mrs. Arthur D. Long, of Baton Rouge, State president of the Women's Auxiliary to the Louisiana State Medical Society, visited the St. Tammany Parish Auxiliary on Wednesday. Mrs. Long had with her her mother, Mrs. Stewart, of El Paso, Texas, and Miss Stevenson of Baton Rouge.

The meeting was held at the home of Dr. and Mrs. Carl Young and was called by Mrs. Young, who is State Counselor for the Sixth District and a Past State President. Mrs. Herbert Cannon, president of the St. Tammany Parish Medical Auxiliary, conducted the meeting. The State president, Mrs. Long, gave an interesting and instructive talk. Attending the meeting from Bogalusa were Mrs. Desporte, Mrs. Fleming and Mrs. Ward, Mrs. John K. Griffith, from Slidell and Mrs. M. C. Wigington of Hammond. Those from Covington attending were Mrs. H. E. Gautreaux, Mrs. B. B. Warren, Mrs. Thomas J. Healy and Mrs. A. L. Saxon. Beautiful camellias from the garden of the hostess decorated the rooms of the home. Following the business session, a delicate plate lunch was served by the hostess. She was assisted by Mrs. A. L. Saxon.

BOOK REVIEWS

Atlas of Surgical Approaches to Bones and Joints: By Toufick Nicola, M. D., F. A. C. S. New York, Macmillan Company, 1945. Pp. 218 illus. Price $5.00.

The line drawings in this book are of excellent character. In addition to depicting accurately the anatomy involved in each of the more common approaches to the bones and joints, the accompanying text helps the reader to find the important structures in the area.

The only criticism that can be offered is that some of the indications and contraindications for

a given incision or approach might have been added. For the student and beginner in the art of surgery this is unfortunate as Dr. Nicola is certainly able to advise in this respect.

This book, however, is highly recommended to all who perform surgical procedures on the extremities as a means of reviewing and refreshing the memory concerning area anatomy.

FRED S. MARSHALL, M. D.

The Chemistry of Anesthesia: By John Adriani, M. D., Springfield, Illinois. Charles C. Thomas, 1946. Pp. 536. Price $7.00.

The scope of this book is much wider than that which might be suggested by the title. As well as a consideration of the chemistry of substances which are commonly used as general anesthetics, attention is given to related compounds having a depressant action on the central nervous system but which for various reasons are unsuitable for practical application. In addition other groups of drugs of interest to the anesthetist are included such as the barbituric acid compounds, the opium alkaloids, the analeptics and drugs affecting the autonomic nervous system. Agents used in the field of local anesthesia are also covered. One omission is that of the curare alkaloids and compounds with similar action which are of recent interest in the field of general anesthesia.

The book is divided into three parts. Part I: Inorganic Chemistry Related to Anesthesia; Part II: Organic Chemistry Related to Anesthesia; Part III: Biochemistry Related to Anesthesia. Part I includes an excellent review of the gas laws, the chemistry of nitrous oxide, carbon dioxide and the gases of air and also a chapter on the absorption of carbon dioxide in rebreathing appliances. In Part II the organic compounds are considered according to chemical groups such as hydrocarbons, alcohols, aldehydes and ethers proceeding to such special classes as the non-volatile hypnotics, opium alkaloids, local anesthetics, and analeptics. In the section on Biochemistry (Part III) one chapter is devoted to proposed mechanisms of narcosis while others are concerned with the effects of anesthesia on the composition of body fluids, liver function, urine formation and general metabolic processes. An extensive bibliography is appended.

Although the author has prepared this book primarily for the anesthesiologist, due to the breadth of subject matter the biochemist, pharmacologist, and internist will find it a source of valuable information.

RALPH G. SMITH, M. D.

Studies on Brazilian Anophelines from the Northeast and Amazon Regions: By O. R. Causey, L. M. Deane, and M. P. Deane. (The American Journal of Hygiene Monographic Series, No. 18.) Baltimore, the Johns Hopkins Press, 1946. viii, 50 p.; 19 pl. Price $1.10.

This small, well-printed volume contains three sections, each with an illustrated key for the identification of anopheline mosquitoes of the northeast and Amazon regions of Brazil, by means of adult female, male genitalic, and larval characteristics, respectively. The first section also presents notes on the malaria vectors, a table for the differentiation of the "tarsimaculatus complex", a table listing 35 species and their geographic distribution in the area, and five plates of line-cut illustrations of wing, leg, palpal, and body characters. The second key is introduced by a short description of male genitalia and of the technic of dissection, includes 34 species, and is illustrated by six half-tone plates, five bearing excellent photomicrographs of diagnostic parts of the genitalia of each species. The key to larvae includes 32 species and is accompanied by descriptions of two larvae (*Anopheles galvaoi* and *A. shannoni*) and a table of the distribution and types of breeding places. Eight plates of line drawings illustrate larval characters.

The authors state that three important malaria vectors—*Anopheles gambiae, aquasalis,* and *darlingi*—are among the 35 species of anopheline mosquitoes found in this region during surveys under the auspices of the Ministry of Education and Health of Brazil, the International Health Division of the Rockefeller Foundation and the Institute of Inter-American Affairs. *A. Gambiae* was introduced accidently from Africa and caused severe epidemics of malaria before it was eradicated in 1940. Due to its high susceptibility to malaria, its house-frequenting habits, and its preference for human blood, it is the most dangerous vector known. *A. darlingi* and *aquasalis* are indigenous vectors, the former the more efficient and wherever present always accompanied by malaria. *Darlingi* is dependent upon high humidity and rainfall, occurs chiefly along large river valleys, and has seasonal fluctuations which account for the characteristic small regional epidemics of malaria in the Amazon Valley. *Aquasalis,* a coastal brackish-water species, is important as a vector only when numerous and causes a low rate of endemic malaria rather than severe rainy-season epidemics like *darlingi.* Another common species, *A. albitarsis,* seems to include biologically different subspecies, only one of which appears to play a secondary role in the transmission of malaria as a result of appropriate habits in some localities. *A. pessoai* likewise is a vector only under certain local conditions. These observations again emphasize the complexity of malaria epidemiology and the need for exact biological knowledge and continued research to unravel the tangled web and to guide efficient and intelligent control measures.

This volume should constitute an excellent tool for the working entomologist and malariologist in

northern Brazil and adjacent areas. It is a sound contribution to our knowledge of a group of insects that is of outstanding importance in public health.

ALBERT MILLER, Ph. D.

Synopsis of Physiology: By Rolland G. Main, Ph. D. St. Louis, C. V. Mosby Co., 1946. Pp. 341. Price $3.50.

This small volume is the outcome of numerous requests for an up to date review of the most recent advances in physiology. The author assumes that his readers are familiar with the rudiments of the subject and so it is approached from the point of view of a text for advanced students and physicians requiring a quick and authoritative reference. There are no references. The author feels these can be ascertained in standard textbooks. It is well indexed and should serve the purpose for which it is intended.

I. L. ROBBINS, M. D.

Principles of Hematology: By Russell L. Haden, M. A., M. D. Philadelphia, Lea & Febiger, 1946. Pp. 366. Price $5.00.

This is a third edition, thoroughly revised. By this time it needs no introduction to the medical public. Changes in the text necessitated by modern advances in hematology have been noted and incorporated in this new edition, as for example, the technic of bone marrow puncture and the study of bone marrow films. However, the last sentence in the preface to the third edition is somewhat puzzling. Thus to quote: "More complicated methods such as the study of the Rh factor have again been intentionally omitted to keep this volume a simple discussion of the fundamental principles of hematology." All authors, editors, teachers kindly take note. The illustrations are good and the format of the book fine.

I. L. ROBBINS, M. D.

Synopsis of Pathology: By William Arnold Douglas Anderson, M. A., M. D., F. A. C. P., 2nd. ed., St. Louis, The C. V. Mosby Company, 1946. Pp. 741, 327 Illus., 15 color pl. Price $6.50.

In the preface to the first edition (1942), Anderson states that his work is "intended to fill a gap which has existed between the very elementary manuals of pathology and the abundant excellent larger textbooks and reference works." It is felt that such a "gap" did exist and that this "Synopsis" has very adequately and effectively filled it. The present second edition is approximately 100 pages longer than the first and contains 23 more text illustrations. Of the total illustrations 74 are new. There are two less color plates in the second edition.

Text material and references have been brought up to date. One is indeed most pleasantly amazed at the conciseness and completeness of so compact a volume. Of course, there are omissions of finer and more technical points, but such omissions are only in accordance with the objective of the book. Special mention must be made of the illustrations. These are abundant and of a most excellent quality. They alone would make this book valuable.

The handy size is also an asset, since it is easily carried. The book would certainly seem to be useful, as suggested by the author, to "the clinician who must maintain familiarity with the foundation sciences of medical practice", as well as to the medical student.

W. H. HARRIS, JR., M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, Inc., New York: Progress In Gynecology by Joe V. Meigs, M. D.

Paul B. Hoeber, Inc., New York: Postgraduate Obstetrics by William F. Mengert, M. D.

Bruce Humphries, Inc., Boston: The Effect of Smallpox on the Destiny of the Amerindian by E. Wagner Stearn, Ph. D. and Allen E. Stearn, Ph. D.

King's Crown Press, Morningside Heights, New York: Motor Disorders in Nervous Diseases by Ernst Herz, M. D. and Tracy J. Putnam, M. D.

Lea & Febiger, Philadelphia: Cushny's Pharmacology and Therapeutics by Arthur Grollman, A. B., Ph. D., M. D., F.A.C.P., and Donald Slaughter, B. S., M. D. The Treatment of Diabetes Mellitus by Elliott P. Joslin, A. M., M. D., Sc. D.

Masson et Cie, Paris, France: L. Tuberculose Renale by Jean Cibert. Le Syndrome de Volkmann by Maurice Cahuzac and Francois Jung.

Oxford University Press, Toronto: Heparin in the Treatment of Thrombosis by J. Erik Jorpes, M. D.

Penguin Books, Inc., New York: The Story of Human Birth by Alan Frank Guttmacher, M. D.

W. B. Saunders Company, Philadelphia: Allergy in Theory and Practice by Robert A. Cooke, M. D., Sc. D., F. A. C. P. Gynecological and Obstetrical Pathology by Emil Novak, A. B., M. D., D. Sc. (Hon. Dublin) F. A. C. S.

Charles C. Thomas, Springfield, Illinois: Office Endocrinology by Robert B. Greenblatt, B. A., M. D., C. M. The Lung by William Snow Miller, M. D., Sc. D.

University of Chicago Press, Chicago: Radical Surgery in Advanced Abdominal Cancer by Alexander Brunschwig, M. D.